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THE SENSORY TRACT IN THE CENTRAL NERVOUS SYSTEM.*

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IT is the object of this essay to determine the course of the tracts which convey general sensations from the surface of the body to the cortex of the brain, and to ascertain in what region of the cortex the sensations are consciously perceived. By the term general sensations it is intended to include tactile sensations, the sensations of pain and temperature, and the muscular sense. In order to determine the course of the tracts it will be necessary to trace the anatomical connection between the skin and the cortex of the brain as far as possible by the various methods at command. In order to ascertain the region of the cortex in which sensations are perceived the physiological experiments which have a bearing upon the subject will be reviewed, and the pathological cases in which sensory symptoms can be connected with localized disease will be cited.

I.—The anatomical connection between the surface of the

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body and the cortex of the brain may be conveniently divided into four parts:

1. The peripheral nervous system, by which sensations reach the spinal cord.

2. The sensory tracts in the spinal cord.

3. The sensory tracts in the medulla, pons, and crura cerebri.

4. The sensory tracts in the brain.

1.—*The peripheral nervous system* requires but a brief mention since its anatomy is a subject remote from the present one. Sensory impressions are transmitted from the surface of the body to the spinal cord along mixed nerves made up of fibres which are partly sensory and partly motor. These nerves after passing between the vertebræ into the spinal canal divide into two divisions, one of which enters the spinal cord upon its antero-lateral surface, and the other of which enters it upon its postero-lateral surface. It was one of the early discoveries of nervous physiology, made independently by Magendie and Sir Charles Bell, that the anterior of these divisions contained all the motor fibres of the nerve, and that the posterior of these divisions contained all the sensory fibres of the nerve. It is, therefore, with the nerve-fibres entering the spinal cord by the posterior nerve-roots that this essay has to deal.

2.—*The sensory tracts in the spinal cord.*—At its point of entrance into the spinal cord each sensory nerve-trunk loses its sheath, and its separate fibres at once diverge in various directions. Two main divisions of the nerve are well marked. The first passes directly into the posterior gray horn. The second passes into the posterior lateral white column, and is the larger of the two.

(1) The nerve fibres entering the posterior gray horn at once diverge from one another. Some of these end in the small cells of the posterior horn. A part of these are lost in

the network of nerve fibres in the substantia gelatinosa. A part of these pass forward to the anterior gray horn of the same side. Others pass over to the gray matter of the other side through the posterior gray commissure. A considerable part pass forward and inward, and then turn upward in a bundle named by Kölliker the longitudinal bundle of the posterior horn, and by Clarke the ascending bundle of Deiters, and having ascended a longer or shorter distance again become horizontal and join gray cells in the posterior horn.

(2) The nerve fibres entering the posterior median white column divide into: (*a*) a small bundle which turns downward, and having passed from one to three centimetres in this direction, enters the posterior gray horn[1]; and (*b*) a large bundle which turns upward, and in its course approaches the antero-lateral angle of the posterior column. This latter bundle sends out its fibres in various directions as it passes upward. Many of these fibres turn outward and enter the posterior gray horn at various levels. The remainder pass upward and are lost. The termination of those entering the gray horn is various. Some end directly in the small cells of the gray horn. Some pass forward to the anterior gray horn of the same side. Some terminate in the groups of cells lying in the posterior lateral part of the anterior horn. Some are lost in the network of Gerlach, in the region of the cells of the column of Clarke, and are doubtless connected with these cells. Many of the fibres pass by way of the posterior gray commissure to the opposite side of the cord, where they end in the gray network, or in the cells of the anterior horn.[2] The various cells in which these fibres terminate send out two or more protoplasmic

[1] Schultze: *Arch. f Psychiatrie*, xiv., 2. Ueber secondären Degeneration im Rückenmarke.

[2] Flechsig: "Leitungsbahn im Gehirn u. Ruckenmark," 1877. Schwalbe: "Neurologie," 1881. Ross: "Diseases of Nervous System," Anat. introduction (1883).

prolongations and these in turn uniting make up a complex network throughout the gray substance of the cord.

Thus all the nerve fibres entering by one nerve-trunk terminate in the gray matter of the cord.[1] The level of their terminal cells is not that of their entrance, but each spinal nerve is connected with successive layers of the gray matter from a point three cm. below its entrance to a point at least eight cm. above and possibly higher.[2]

From the cells in the gray matter and from the network of fibres new nerve fibres arise which pass backward to the posterior median column, and form it. Some of these fibres come from the inner side of the posterior horns and from the network near the Clarke column of cells, and pass through the posterior lateral column to reach the posterior median column. Others come from the posterior commissure of the gray substance, and turning directly backward pass along the sides of the septum posterius and thus reach the median column.[2] This posterior median column is made up entirely of long fibres passing up to the medulla and conveying sensations from the lower extremities.[3] The posterior lateral column below the cervical region contains chiefly short fibres passing from the nerve-trunks to the gray matter as already described; and possibly short fibres between adjacent segments of the cord. In the cervical region it also contains long fibres which pass up to the medulla—and which probably convey sensations from the upper extremities.[3] The direct cerebellar columns, lying in the periphery of the lateral columns are made up of fibres which arise from the cells of the columns of Clarke—cells which are considered the terminal stations of some of the sensory fibres entering the posterior horn. These

[1] Hermann's "Handbuch. d. Physiol." Eckhard. P. 159.

[2] Flechsig: "Leitungsbahn im Gehirn und Ruckenmark," p. 311.

[3] Schultze: *Arch. f. Psychiatrie*, xiv., 2. Ueber secondären Degeneration. Flechsig: Plan des mensch. Gehirns.

columns were thought to convey sensory impulses by Meynert,[1] and the fact of their origin from sensory cells, proven by Flechsig,[2] is supposed to confirm this hypothesis, which is further strengthened by the fact that they degenerate upward (centripetally) after lesions in the cord.

It has been recently asserted that some of the sensory fibres in the cord pass upward in the portion of the lateral columns which lie between the gray matter of the cord and the direct cerebellar columns. (Pyramidenbahn and Seitenstrang reste.) This hypothesis is not proven, and the fact that in descending degeneration involving these columns after a lesion of the brain or upper cord, as well as in primary lateral sclerosis which affects these columns in their entire extent, all sensory symptoms are wanting, seems to oppose the hypothesis.

Anatomical study therefore indicates that in the spinal cord the sensory tracts lie in :

1. The gray matter of the cord ;
2. The posterior median and posterior lateral columns ;
3. The direct cerebellar columns.

Physiological experiment and pathological study have shown that sensory impressions pass immediately, after their entrance into the cord, to its opposite side, at least partially if not wholly.[3] The decussation of the fibres conveying sensations of pain and temperature along any nerve occurs at a lower level than that of the fibres conveying tactile sensations.[4] The fact that the sensations of pain and temperature may be lost while those of tactile sensations are unaffected,[5] and the fact that tactile sensation may be impaired while sensations of pain and temperature

[1] Meynert : *Arch. f. Psych.*, iv.

[2] Flechsig : " Leitungsbahn," p. 299.

[3] Hermann's " Handbuch d. Physiol.," p. 167.

[4] Köbner, *Arch. f. klin. Med.*, xix., 169, art. on Spinal Hemiplegia—forty-eight cases.

[5] Bernhardt, *Berlin. klin. Wochenschr.*, Jan. 24, 1884.

are normal,[1] indicates that these two classes of sensation are transmitted along different tracts in the spinal cord in man. In animals sensations of pain and temperature are transmitted upward through the gray matter of the cord; and tactile sensations are sent along the posterior white columns.[2] It is probable that the same is true in man, but this is not yet proven, as it is possible that in disease of the cord in man sensations may pass upward through the gray matter, which would normally go along the posterior columns.[3]

It may therefore be stated that the various functions of the various sensory tracts is not absolutely determined in man. It is known that a unilateral lesion of the cord in man produces a loss of muscular sense on the side of the lesion, and a loss of the sensations of touch, pain, and temperature on the side opposite to the lesion.[4] It is also known that sclerosis of the posterior lateral columns, which involves the fibres of the sensory nerves entering the cord through them, produces disturbances of the sensations of touch, pain, and temperature, as well as a marked disturbance of the muscular sense. Such a sclerosis is followed after a time by secondary scleroses in the posterior median and direct cerebellar columns, but it is as yet impossible to distinguish the symptoms due to each of these processes. After a transverse myelitis from any cause a secondary degeneration upward of the sensory tract occurs, the process advancing up to the medulla in the posterior median and direct cerebellar columns and for several centimetres up-

[1] Schüppel, *Arch. f. Psych.*, ix.

[2] Schiff, "Physiologie"; confirmed by M. Foster and Dalton.

[3] Wundt, "Physio-Psychol.," pp. 107–110; also Hammond, Case of Allochiria, N. Y. Neuro. Soc. Trans., Jan., 1883; and cases of allochiria in *Brain*, vol. iv., No. 2, vol. v., No. 3.

[4] Brown-Séquard, *Arch. de Physiol.*, 1868; Köbner, *Arch. f. klin. Med.*, xix., where forty-eight cases with autopsies are collected. Brown-Séquard considered that the muscular sense was transmitted through the motor columns of the cord, but more recent investigation has demonstrated the existence of sensory nerves from the muscles which enter the posterior columns, and are therefore in connection with the sensory tracts.

ward in the posterior lateral columns,[1] and when this is total all sensations are cut off from below. When the process is only partial, however, sensation is but slightly affected, if at all; though whether this lack of symptoms is due to the escape of some white fibres in the degenerated columns, or to a vicarious action of the gray matter, is undecided, although the former is probably the case.

None of the pathological processes in the spinal cord which affect the sensory tracts afford a basis for distinguishing between the functions of these various tracts. The only conclusions possible in the present state of knowledge are:

(1) In the spinal cord sensory impressions are transmitted through the posterior median and posterior lateral and direct cerebellar white columns, and through the gray matter.

(2) All sensory impressions except those of muscular sense decussate in the cord soon after their entrance to a great degree, if not wholly.

3.—*The sensory tracts in the medulla, pons, and crura cerebri.* On reaching the medulla the sensory tracts of the cord undergo changes of direction and division which are difficult to trace. It is impossible to rely solely upon any single method of determining their course. A number of methods, however, are at present used to trace the direction of a nerve-tract. These are as follows:

(1) By means of a series of thin continuous sections, sagittal, horizontal, and vertical, the continuity of fibres can be followed.

(2) As the medullary sheaths of the fibres of each tract develop in the fœtus at various times, their presence or absence in a definite area, in fœtuses of various ages, affords valuable information regarding the course of the tracts.[2]

[1] Schultze, *Arch. f. Psychiatrie*, xiv., 2, Über secondären Degeneration; and Homen, *Virchow's Arch.*, Sept., 1882.

[2] Known as Flechsig's method.

(3) If a definite tract be divided in a new-born animal, and it survives the operation, the parts connected with that tract fail to develop as the animal grows, and hence a *post-mortem* examination of the full-grown animal will reveal an atrophy in the course of the tract, formerly divided, whose course it is desired to trace.[1]

(4) A local focus of disease in any nerve-tract is followed in the course of a few weeks by a process of degeneration in that tract, which probably proceeds in the direction in which normal nerve impulses are sent. Hence, some time after a lesion, the tract leading from that lesion may be distinguished from all other tracts by the condition of degeneration.

(5) By collecting a large number of cases of local lesion limited to a small area of the nervous system, and ascertaining the symptoms common to all these cases, valuable positive information regarding the function of the area involved by the lesion is afforded. The evidence thus obtained may be tested by a second collection of cases of local lesion involving all other parts of the nervous system excepting the area concerned in the first set of cases, and observing in these the function which remains unaffected. If the facts afforded by the positive cases are substantiated by those obtained from the negative cases, they may be considered as established upon a firm basis.

It is by the use of all these methods, and by a selection of the common facts established by them, that we shall attempt to determine the course of the sensory tracts in the medulla, pons, and crus.

(1) The continuity of the sensory tracts of the cord, with certain parts of the medulla, is easily ascertained, and is not a matter of dispute. All authorities admit, that the posterior median column (the column of Goll) terminates

[1] Known as Gudden's method.

in the funiculus gracilis of the medulla, and its fibres enter the cells of the nucleus gracilis. The posterior lateral column (the column of Burdach) terminates in the funiculus cuneatus and its nucleus, which lies just external to the funiculus gracilis. The direct cerebellar column of the cord passes directly to the cerebellum, by the way of the corpus restiforme of the medulla, the column lying just external to the funiculus cuneatus. The gray matter of the cord undergoes a change of form and of location in becoming continuous with that of the medulla. Each posterior horn becomes greater in area at its extremity, and smaller in area at its junction with the central gray matter, and finally its extremity is separated from the central gray matter by a set of white fibres which come from the nucleus cuneatus and nucleus gracilis, and pass inward toward the median line. While this change of form is proceeding, the location of the posterior horn is also changed. It is displaced laterally by the constantly increasing size of the parts lying between it and the median fissure, and later by the gradual opening out of the central canal into the fourth ventricle by means of the separation of the sides of that fissure. Thus it comes to lie in the lateral part of the medulla, a little posterior to a horizontal line through its centre. The microscopic appearance also changes. Instead of a compact mass of small gray cells in the cord, lying in a fine network of fibres, the sensory gray of the medulla presents the appearance of scattered gray cells lying in a coarse network of fibres. These fibres are partly protoplasmic prolongations of cells, and partly distinct white fibres passing in all directions. The appearance of this area in the medulla has been described by the name which it bears—viz.: the formatio reticularis. In the lower part of the medulla, therefore, the sensory tracts on each side consist of three gray col-

umns—the funiculus gracilis, the funiculus cuneatus, and the formatio reticularis; and a single white tract—the direct cerebellar column. The latter passes out of the medulla by the inferior peduncle of the cerebellum, and terminates in the cortex and central gray matter of the vermiform lobe of the cerebellum. It does not enter into the description of the sensory tracts above this level, and will therefore be traced separately further on. The course of the sensory impulses from the three gray columns upward through the medulla and pons is still undetermined, since irreconcilable differences exist between the various anatomists who have studied the subject. The following views have been advanced by different authorities:

MEYNERT [1] traces fibres from each nucleus gracilis and nucleus cuneatus (1) to the olivary body of the same side, where fibres arise which cross to the opposite side and go to the cerebellum through the corpus restiforme; (2) through the sensory decussation to the opposite side of the medulla, where they turn upward in the interolivary tract, lying adjacent to the raphé and dorsad to the pyramidal tract. These fibres pass up through the pons, lying just dorsad to the pyramidal tract, and in the crus they divide, some ending in the substantia nigra, and some going to the external third of the longitudinal bundles of the pes of the crus, and thence to the posterior part of the internal capsule. He states that the outer part of the formatio reticularis, in which lie the sensory roots and cells of the V., and VIII., and IX. nerves, is homologous to and a continuation of the substantia gelatinosa of the posterior gray horns of the cord below, and is continuous above with the formatio reticularis lying external to the red nucleus of the tegmentum of the crus, whence fibres ascend to the posterior part of the internal capsule, and to the optic thalamus.

FLECHSIG [2] traces (1) fibres from the nucleus gracilis through the upper decussation to the opposite interolivary tract, in which they ascend and then pass to the lateral part of the lemniscus, in which they go upward between the corp. quad. and the red

[1] Stricker's "Handbook of Histology," Sydenham edition, vol ii., p. 525. *Arch. f. Psych.*, iv., p. 405.

[2] Flechsig: "Leitungsbahn im Gehirn und Ruckenmark," 1876. *Arch. f. Heilkunde*, xviii., p. 280, 1877. "Plan des menschlichen Gehirns," 1883, pp. 21, 22. *Arch. für Micro. Anat.*, xix., p. 60, 1881.

nucleus to the posterior third of the internal capsule. (2) Fibres from the nucleus cuneatus (*a*) to the olivary body of the same side, which is connected (*α*) with the lemniscus of the same side, and (*β*) with the cerebellum of the opposite side; (*b*) to the formatio reticularis of the same, and possibly to the opposite side, whence fibres pass both to the optic thalamus and to the cortex through the internal capsule. He considers the lemniscus and the formatio reticularis as the direct continuation of the posterior columns of the cord, and opposes the view which connects these columns with the cerebellum. With the formatio reticularis are also continuous the gray matter of the posterior horns of the cord and the white fibres lying adjacent to it (Seitenstrang reste).

AEBY [1] traces (1) the fibres from the nucleus gracilis through the upper decussation into the interolivary tract, thence through the lemniscus to the optic thalamus; (2) the fibres from the nucleus cuneatus to the olivary body of the same side, thence to the corpus restiforme of the opposite side, and in it to the cerebellum, whence they issue again in its superior peduncle, and decussating a second time below the red nucleus end in it or in the optic thalamus above.

ROLLER [2] considers the interolivary tract and its continuation upward in the lemniscus as the chief sensory tract of the medulla and pons, and traces to it fibres from both the nucleus gracilis and nucleus cuneatus of the opposite side, and fibres from the olivary body of the same side, with which both the nuclei of the same and of the opposite side are joined. The lemniscus does not decussate in the crus, but passes directly to the posterior part of the internal capsule, and to the optic thalamus.

HENLE [3] describes a second decussation of the lemniscus in the crus in the roof of the aqueduct of Sylvius, a view with which the description of Wernicke is in accord. Henle does not attempt to trace each tract of the cord to the brain.[4] He states that the greater part of the posterior columns of the cord join the anterior column of the medulla and thus ascend, a position in which he stands alone, and which has never been confirmed.

SCHWALBE [5] says that a connection of the nucleus gracilis and

[1] Aeby: "Schema des Faserverlaufes im Gehirn u. Ruckenm." Bern, 1883.

[2] Roller: "Die Schleife." *Arch. für Micro. Anat.*, xix., 2, p. 303.

[3] Henle: "Handbuch der Nervenlehre," 1879, p. 272.

[4] *L. c.*, p. 329.

[5] Schwalbe: "Lehrbuch der Neurologie," 1881, p. 633.

nucleus cuneatus, with the formatio reticularis and olivary body, though probable is not proved, and that their connection with the cerebellum is very improbable. He considers the formatio reticularis as the continuation of the seiten strangreste, and traces its fibres to the optic thalamus. He does not trace a sensory tract directly from the cord to the brain, but confines himself to a review of other anatomists, preferring to leave the question undecided.

WERNICKE [1] has given the subject most careful study, and come to the following conclusions. Since the sensory impulses cross the median line in the cord soon after their entrance, and since lesions of the cord or of the internal capsule produce hemianæsthesia of the opposite side, the sensory tract in one side of the cord must be continuous with the internal capsule of the same side. Therefore there is either no sensory decussation in the medulla or there is a double sensory decussation between the spinal cord and the internal capsule. If the first alternative is adopted the sensory tract must lie in the medulla in the external lateral part of the formatio reticularis. This conclusion is reached by a process of exclusion, the function and connection of all other parts being distinctly non-sensory, except the lemniscus, which, however, cannot be included as it decussates. But Wernicke has observed a case of tubercle of the pons,[2] situated just in the external lateral part of the formatio reticularis in which no anæsthesia of the parts below was produced. He therefore rejects this alternative, and adopts the second one, according to which a double decussation of the sensory tract occurs between the cord and capsule. This double decussation he traces as follows : [3]

A. Fibres from the nucleus gracilis cross in the upper or sensory decussation to the interolviary tract and lemniscus, ascend in it to the roof of the aqueduct of Sylvius beneath the corpora quadrigemina, where they recross (in the decussation described by Henle), and then pass up into the internal capsule, and to the optic thalamus.

B. 1. Fibres from the direct cerebellar tract of the cord pass along the inferior cerebellar peduncle to the vermiform lobe of the cerebellum where their first decussation occurs.

2. Fibres from the nucleus gracilis pass along the inferior cerebellar peduncle of the same side to the vermiform lobe where their first decussation occurs.

[1] Wernicke : " Lehrbuch der Gehirnkrankheiten." Bd I., 1881.

[2] *Arch. f. Psych.*, vii.

[3] I reverse the order in which he states the facts, in order to trace the tracts from below upward in the direction in which they transmit impulses.

3. Fibres from the nucleus cuneatus pass to the olivary body of the same side, thence by the fibræ arcuatæ to the inferior pednucle of the opposite side, thus reaching the dentate nucleus of the cerebellum.

The fibres then issue from the cerebellum by two paths : (1) by the middle peduncle, when they recross in the pons and turn upward in its anterior part to pass into the outer third of the pes of the crus, and thence reach the internal capsule ; (2) by the superior peduncle, when they recross in its decussation below the red nucleus, and, passing around or through this, reach the internal capsule.

The last authority to be mentioned is SPITZKA,[1] whose description of the sensory tract agrees in some points with that of Meynert and in some with that of Wernicke. He holds that the fibres from the nucleus gracilis pass through the sensory decussation of Meynert to the interolivary tract of the opposite side, and thence in the stratum intermedium (a term which Spitzka applies to the longitudinal fibres of the pons lying just posterior to the pyramidal tracts, and which is the same as Henle's Bundle vom Fuss zu Haube, and which is continuous with Meynert's stratum intermedium at the level of the substantia nigra of Sömmering) to the posterior part of the internal capsule, thus forming a direct sensory tract to the cerebrum.[2] He holds that the fibres from the nucleus cuneatus divide into a larger and a smaller set of fibres. The larger set decussates through the olivary bodies, and thence passes to the cerebellum by way of the restiform column. The smaller set does not decussate, but passes to the inner portion of the restiform column of the same side, forming the inner division of the inferior peduncle of the cerebellum, and terminating in the nucleus fastigii.

The direct cerebellar column joins the restiform column, and thus represents a direct uninterrupted communication between the sensory periphery and the hemispheres of the cerebellum. The restiform column of each side is therefore made up of (1) the direct cerebellar column of the same side ; (2) a part of the column of Burdach of the opposite side, whose fibres have passed through

[1] The Relations of the Cerebellum, *Alienist and Neurologist*, Jan., 1884. Case of Pons Lesion, *Amer. Jour. of Neurology*, Feb., 1884.

[2] Spitzka does not seem to notice what Wernicke pointed out, that, if such were the course of the sensory tract conveying tactile sensations, a lesion of the internal capsule would produce anæsthesia on the *same* side of the body as the lesion. For the sensory impulses along such a tract would have crossed once in the cord and a second time in the medulla, thus regaining the side they came from.

the olivary bodies ; (3) a part of the column of Burdach of the same side. The restiform column goes to the cortex of the cerebellar hemisphere, and a small portion of it to the nucleus dentatus of the cerebellum. From these parts new tracts issue which convey sensory impulses to the cerebrum. The first of these tracts is the middle peduncle of the cerebellum, in which fibres pass from the hemisphere of the cerebellum to the pons of the opposite side, end in the gray matter of the pons, and thence go up by new longitudinal fibres in the pons, which pass into the pes, and thus to the internal capsule. (This corresponds to Wernicke's first recrossing tract.) The second of the tracts passes from the nucleus dentatus of the cerebellum along the superior peduncle to the red nucleus of the opposite side, and thus into the internal capsule. (This corresponds to Wernicke's second recrossing tract.) Spitzka holds that the cerebellum is a special cerebral segment for all the sensory tracts, into which they are temporarily deflected on their way to the cerebrum. He therefore favors the view of Wernicke as to the existence of a double decussation between the cord and the capsule for all fibres excepting those of the column of Goll. Spitzka, therefore, traces two distinct sensory tracts from the cord to the brain. One of these goes directly through the interolivary tract, stratum intermedium, crus, and internal capsule to the cortex. The other is deflected to the cerebellum, and, having decussated twice between the lower level of the medulla and the level of the red nucleus, reaches the internal capsule and joins its fellow. The first of these corresponds to Meynert's tract. The second of these corresponds to Wernicke's tract.

It is evident that the different views here stated cannot be reconciled. It is also evident that the method of tracing fibres followed by these anatomists is inadequate to decide between their differences. The actual course of the sensory tracts must be traced by the aid of other methods.

(2) The method of Flechsig gives little satisfactory information as to the sensory tracts, as he himself admits, and the conclusions reached by him are based largely upon the results of pathological study. They have been already stated.

(3) The method of Gudden has been pursued by but one experimenter,[1] and but one of his experiments was successful. This, however, affords important imformation and is therefore stated in full.[2] Von Monakow of St. Pirminsburg divided one half of the spinal cord just below the medulla in a new-born rabbit. Six months afterward the animal was killed. The following parts above the place of division were found atrophied: the posterior median column with the nucleus gracilis except in its median half which had not been involved in the division; the posterior lateral column with the nucleus cuneatus; the direct cerebellar column and its tract in the corpus restiforme; the formatio reticularis in its external two thirds lying internal to the ascending root of the fifth nerve, from the medulla as far up as to the anterior corpus quadrigemnum; the lateral part of the lemniscus; the fibres connecting the nuclei gracilis et cuneatus with the olivary body (bogenfasern); the centre of Deiters or outer acoustic centre, on the side of division. On the side opposite the division the olivary body and the lemniscus were partly atrophied. The atrophy could not be followed up to the optic thalamus or internal capsule, hence v. Monakow concludes that no direct sensory tracts exist from the cord to the brain, but that all impulses are sent to gray centres in the medulla and pons, and thence are transmitted indirectly by new fibres upward. The paths of sensory impulses as determined by his experiment would be the formatio reticularis and interolivary tract in the medulla and the lemniscus and formatio reticularis in the pons and crura, and the corpus restiforme to the cerebellum. But as the atrophy in the corpus restiforme involved only the direct cerebellar columns all the way to the cerebellum, and affected its inner

[1] That is for the purpose of determining the course of the sensory tracts.

[2] Monakow: "Exper. Beitrag zur Kenntniss des corp. Restiform." *Arch. of Psych.*, xiv., 3, Nov., 1883.

third only as far as the Deiters centre which lies in the medulla, he considers the transmission to the cerebellum of sensory impulses, except by the direct cerebellar columns, a matter of much doubt. These results of his investigation agree with the statement of Flechsig in the most important particulars and oppose distinctly the statement of Wernicke as to a double decussation through sensory tracts passing to the cerebellum.

Closely allied to this method of Gudden and in some respects in correspondence with it is a method of investigating the course of sensory tracts in infants born with defective brains. If (as Flechsig teaches) the tracts between brain and spinal cord develop in the direction in which they convey impulses, it would be possible to distinguish between centripetal and centrifugal fibres definitely in the nervous system of an infant born with a nervous system deficient either as to brain or as to cord.

The subject of microcephalic brains is one to which hitherto little attention has been given, probably because of their rarity, and also because their importance for anatomical purposes has not been appreciated. I have been able to find but one well recorded case of a microcephalic brain.[1] I have been fortunate enough to secure such a brain, through the kindness of Dr. Northrop, Pathologist of the New York Foundling Asylum, and its examination has been the original motive for this essay.

The following is the description of this brain.[2] The spinal cord below the second cerv. vert. was unfortunately not removed.

[1] This is the case described by Rohon, and published in pamphlet form by him. My thanks are due to Dr. Spitzka for calling my attention to, and for lending me, this pamphlet which was sent to him by the author.

[2] It may be as well to state that this infant presented by the breech, was born alive, and lived one week. It took nourishment and cried like a normal infant, but made no voluntary movements and seemed incapable of perceiving sensations. It slept much of the time, waking only to nurse. It did not attract attention by any peculiar symptoms, and no further history of it was taken.

Microcephalic Infant who Lived Seven Days.

Autopsy.—Head : On opening the head by means of incisions along the sutures a large amount of serum was evacuated. The two parietal bones were removed together. It was then found that there was a marked deficiency in the development of the brain, there being no evidence of cerebral tissue in the anterior and middle cranial fossæ or above the tentorium cerebelli. The *dura mater* lined the cranial cavity but presented at several points an abnormal appearance. Anteriorly, in the median line where two layers should been reflected from the sides of the groove to form the falx cerebri but one layer was present springing from the left margin of the groove. The right layer was deficient for a space of two inches anteriorly, so that at this place the falx cerebri was deficient upon the right side, and a probe could be thrust forward into a cul-de-sac (the foramen cæcum) at its anterior extremity. The falx was normal in its formation and attachments posterior to the coronal suture but its margin hung free in the cranial cavity, there being no brain tissue on either side. The longitudinal sinus in the falx was closed anteriorly opposite the coronal suture so that it did not open into the cranial cavity through the deficiency above described. About three quarters of an inch to the left of the falx, and extending parallel with it, arising from the inner surface of the frontal bone, and from the left parietal bone, was an abnormal reduplication of the dura mater similar in appearance to the falx but less extensive. This layer of dura was attached posteriorly to the margin of the left petrous bone, anteriorly to the left posterior clinoid process of the sphenoid bone. It was therefore stretched across the left anterior and middle fossæ and divided the space into two parts, the outer of which was twice the size of the inner. Contained within the layers of this process of the dura and situated near its free margin about half way from the inner surface of the frontal bone to the apex of the petrous bone was a small mass of tissue oval in shape, one inch in antero-posterior and vertical diameters and half an inch in thickness, which had the gross and microscopic appearance of cerebral substance. It had no connection with the cerebral tissue at the base and must have developed independently. It was the only evidence of an attempt at the formation of cerebral lobes. As it could have developed from the prosencephalon only, and as no other tissue was present which had developed from that portion of the cerebral vesicle, it seemed probable that its presence indicated that the prosen-

cephalon was torn away from the thalamencephalon at a very early stage of fœtal life and had therefore failed to develop excepting to this rudimentary degree. On the right side a fold of dura mater extended across the middle cranial fossa. It arose from the posterior margin of the lesser wing of the sphenoid from the clinoid processes, and from the margin of the petrous portion of the temporal bone. It did not reach as a whole the inner surface of the squamous portion of the temporal bone but was held out toward, and partly attached to that bone by six fibrous bands which radiated from its free margin. There was no similar fold on the left side. Other parts of the dura, viz. : the parietal layers and the sinuses, were normal.

The mass of cerebral tissue present was surrounded by a thick *pia mater* containing many *blood-vessels*. A plexus of these vessels lay upon its upper surface. Upon its lower surface the distribution of the vessels was abnormal. The vertebral arteries joined to form a basilar artery, which lay upon the pons, but was not straight, but was thrown into numerous curves. This basilar artery divided into two arteries, each of which passed outward and forward, giving off numerous vessels to the cerebellum, and to the plexus already mentioned, and joined at their anterior terminations the internal carotid arteries. There were no anterior cerebral or anterior communicating arteries, and hence the circle of Willis was not complete.

The cerebral mass which was present is represented in its exact size in figures 1 and 2. The *upper surface* presented the appearance of a normal, but small, cerebellum and medulla, while anterior to the vermis cerebelli a mass of gray matter was seen not presenting an appearance similar to the corpora quadrigemina or optic thalamus, but consisting of a mass of irregular gray substance, continuous with a second mass, the latter having an appearance of two symmetrical oval bodies with smooth surface separated by a shallow fissure. This gray mass was very soft, and in the process of extraction it was injured upon the right side, and was detached from the pons upon both sides.

The cord being cut through, the medulla and cerebellum were lifted carefully, and the entire cerebral mass was removed from behind forward. All the cranial nerves from the xii. to the iii. were divided in turn, none being deficient. From the thick mesh of pia mater and connective tissue lying beneath the irregular gray mass anterior to the pons, the optic tracts, chiasm, and nerves were easily distinguishable, but the optic nerves were smaller than

in a normal brain of like age. There was no appearance of lobulation, and no fissures were seen in the gray mass from which the optic tracts issued to form the chiasm. The tracts could not be traced outward, but sank directly into the mass as represented in fig. 2. The *lower surface* of the brain presented a number of abnormal features. The *medulla* below the level of the olivary bodies was small, and the anterior pyramids were wholly wanting. The olivary bodies were abnormally large and lay adjacent to one another, being separated by a deep fissure. No fibræ arcuatæ were visible. The *pons* presented a normal appearance externally. The longitudinal fibres characteristic of the *crura cerebri* were wanting, and the level of the mass of gray matter, whose surface was rough, which extended forward from the anterior margin of the pons was much below that of the normal level of the crura. From this mass of gray matter the iii. nerves issued directly, and were larger in size than in a normal brain.

The *cerebellum* was somewhat smaller than normal, but appeared to be perfectly developed in all respects; the normal lobes being well defined by the normal fissures. It measured 7.0 by 3 by 1.6 cm. The gray mass lying anterior to the pons presented two anterior prolongations, which ceased abruptly at a a point 1 mm. from the mass.

The entire cerebral mass was put in Müller's fluid to harden, and at the end of six weeks was placed in alcohol.

It was evident from the parts of the cerebrum missing, that the prosencephalon had failed to develop, thus causing an absence of the entire cerebral hemispheres, the olfactory bulb and nerve, the corpus callosum, the corpora striata, the anterior commissure, and the fornix.

The thalamencephalon, or posterior half of the anterior primary vesicle, had developed to some extent, as was evident from the two masses of gray matter from which the optic tracts and nerves arose, but its development was not normal. For the purpose of accurate comparison the normal brain of a child one week old was obtained and hardened, and horizontal sections were then made with a Thoma microtome through the medulla, pons, and crura, both of the normal and of the microcephalic brain. The sections made were from 20 to 30 μ thick, those in the crura being necessarily thicker than those in the medulla and lower pons, on account of the brittle nature of the tissue. Every set of ten successive sections was given a number, and one or two of each set of these sections were stained and mounted. Weigert's method of

staining with acid fuchsin[1] was used, and also the method with ammoniac-carmine. From the mounted sections those were selected for drawing which demonstrated the course of the various tracts and the presence or absence of the various nuclei and objects of interest. It was considered needless to draw all the sections made. Those which are given are drawings of sections at intervals of about 1 cm. from the first cervical nerve upward.[2]

In the spinal cord at the level of the first cervical nerve (fig. 3) it will be noticed that the postero-lateral columns are absent and that in consequence the shape of the cord is abnormal. The anterior gray horns are small, but their cells present a normal appearance. The anterior fissure is deep at the normal position of the pyramidal decussation. The posterior columns, the posterior gray matter, and the caput cornu on both sides are normal.

In the medulla, at the level of the beginning of the nuclei gracilis and cuneatus, the first decussating fibres appear; at the same level, the ascending root of the fifth nerve become distinct. The contrast between the large number of decussating fibres in the normal, and the small number in the abnormal specimens, was marked. The angle at which these fibres decussate was greater (*i. e.*, more obtuse) than that at which the fibres of the pyramidal tracts in the normal specimen met, thus indicating that they are independent of any pyramidal fibres. In the next figure, fig. 4, the origin of these decussating fibres is seen to be from the nuclei gracilis and cuneatus. *The existence of this decussation, in a specimen in which pyramidal tracts are wholly absent, proves the independent existence of the sensory decussation.* It is noticeable that the number of decussating fibres is small, the decussation occupying a small area in all the specimens through this series, and lying deep within the medulla at the bottom of the anterior fissure, and just anterior to the central canal. At the centre of the decussation, where its fibres are most numerous, the area occupied is but one third of that occupied by the decussation at this point in the normal specimen. It is, therefore, evident that in a normal medulla the decussation of motor and sensory fibres takes place, at least in part, at about the same area, and that the pyramidal and sensory decussations do not lie entirely separate, the one below the other. The absence of pyramidal tracts and decussation influences the shape of the medulla to a marked degree,

[1] See Weigert's article, *Centralbl. f. med. Wissenschr.*, 1882, Nos. 40 and 42.

[2] Eighteen drawings with the sections accompanied the essay. Only those which are necessary are given here.

and also the arrangement of the gray matter; the anterior horns not being cut off from the central gray matter in the abnormal specimen until the sensory decussation is fully developed. There is little difference between the normal and abnormal specimen as far as the development of the nuclei gracilis and cuneatus, the nuclei cuniati accessores, the formatio reticularis, and the fifth nerve are concerned. In the sections just above fig. 4, the numerous fibres passing to the olivary body of the *same* side from both nuclei, were clearly seen. In fig. 5, the abnormally large size of the olivary body in the abnormal specimen is already evident, and a large number of fibres are seen issuing from the large olivary body and passing toward the raphé. Many of these can be followed across the raphé into the formatio reticularis of the opposite side, where they are lost as they turn toward the restiform body. The inner olivary nucleus is smaller in the abnormal specimen, and is seen to be circular; its cells are of the same size and character as those of the olivary body. The outer olivary nucleus is absent in the abnormal specimens.

The interolivary tract appears to be much more narrow, and has a shorter antero-posterior (ventro-dorsad) diameter than in the normal specimen; this is evident at all levels, but especially in section fig. 5. The small size of the interolivary tract in this brain supports the assertion of Flechsig. According to Flechsig, the normal interolivary tract consists of two parts. The smaller of these is the continuation upward of sensory fibres which have decussated in the sensory decussation. It increases in size from below upward, in the reverse ratio to that in which the nuclei gracilis and cuneatus decrease in size,—that is, as these nuclei send fibres upward to the opposite interolivary tract, they grow smaller and it grows larger. The larger of the two parts of the interolivary tract is a continuation of that part of the lemniscus which comes from the lenticular nucleus through its pillar (the Linsenkernschlinge). In this microcephalic brain there was no lenticular nucleus; the lemniscus was small, and this second or larger part of the interolivary tract was absent. There were present nuclei gracilis and cuneatus, and the smaller part of the interolivary tract was present. *It is, therefore, evident that a portion of the interolivary tract develops from below upward, and is in relation with the nuclei gracilis and cuneati.* And, further, that a portion of the interolivary tract develops from above downward, and being in relation with the nucleus lenticularis is absent when it is absent.

The raphé in the abnormal specimen is deficient in the number of its antero-posterior (ventro-dorsad) fibres. This is to be brought into connection with the deficiency of the pyramidal tracts, by means of which the central cortex is joined with the cranial nerve nuclei. It has been supposed that fibres leave the pyramidal tracts at various levels in the pons and medulla, and, turning backward, pass along the raphé to the cranial nerve nuclei. The absence of these fibres taken in connection with the absence of the pyramids proves this hypothesis to be well founded.

There is a marked difference between the abnormal specimen and a normal one in the number of decussating fibres in the anterior (ventrad) part of the raphé. This is doubtless due to the same cause as the deficiency of fibres between the pyramids and the cranial nerve nuclei, and may be considered as evidence that the fibres from the pyramids to the nuclei not only pass in the raphé, but also decussate in the raphé soon after entering it, and at a point nearer to the anterior (ventrad) than to the posterior (dorsad) surface of the pons and medulla.

It is to be noted that the shape, size, and appearance of the gray matter on the floor of the fourth ventricle, of the cranial nerve nuclei, and of the roots of the cranial nerves, of the ascending root of the fifth nerve, of the solitary bundle, and of the formatio reticularis, do not differ at all from those of a normal brain. Although to external appearance the medulla was apparently deficient in arciform fibres, the sections show that these are present in about normal numbers.

The deformity in the general shape of sections through the medulla is due to the absence of the pyramid, to the large size of the olivary bodies, and to the existence of a deep fissure between them.

In the pons (figs. 6 and 7) the most noticeable feature of the specimens is the total absence of the *longitudinal fibres* in the ventral half. This half of the pons is made up of transverse fibres coming from the cerebellar hemispheres and of gray nuclei which are scattered irregularly between the transverse fibres, and which cannot be separated into superficial and deep layers as in the normal specimen. The absence of the longitudinal fibres diminishes the size of the pons, and also its shape, the ventral half being narrower in ratio to the dorsal half than in the normal specimen.

The same comparative deficiency of fibres in the *raphé*, both ventro-dorsad and decussating, which was found in the medulla is evident in the pons, and confirms the supposition that these fibres

have a relation to the longitudinal fibres of the pons, and join these with the cranial nerve nuclei.

In a normal pons the *lemniscus* is a noticeable feature, being made up of the fibres from the interolivary tract, which change their relative position as they ascend, turning ventrad and laterad; so that from occupying a rectangular area adjacent to the raphé with its long diameter parallel to the raphé in the lower part of the pons, it comes to occupy an oval area adjacent to the pyramidal tracts with its long diameter at right angles to the raphé in the upper part of the pons. The position of the lemniscus in the abnormal specimen is normal, but its size is small, and the area occupied by its fibres is about one half that in the normal specimen. This corresponds to the deficiency of fibres in the interolivary tract which has been noticed. The fibres of the lemniscus which are deficient are those which lie most internally in the normal lemniscus, the portion which, according to Wernicke, can be traced to the linsenkernschlinge, a formation which is absent in this specimen. As the lemniscus is traced upward through the pons its fibres become more and more deflected away from the median line, so that at the upper level of the pons (fig. 7) they are seen to approach the lateral boundary of the dorsal half and to be curving upward (dorsad).

It is evident, therefore, that a continuous tract can be traced from the nuclei gracilis and cuneatus across the median line, through the interolivary tract into the lemniscus, and upward into the tegmentum of the crus cerebri, where it lies in the external lateral part of that body.

The *formatio reticularis* presents a normal appearance in the abnormal specimen throughout the pons.

The *posterior longitudinal bundle*, whose fibres in the medulla could not be accurately distinguished from those of the interolivary tract, is well seen in all the sections through the pons, and presents a normal appearance both as to size and as to situation.

The nuclei upon the floor of the fourth ventricle occur in their regular order and position and present nothing worthy of remark. The ascending root of the trigeminus increasing in size as it ascends through the lower half of the pons, meets the descending root at the junction of the upper and middle thirds, and being joined by the motor fibres from the motor nucleus the nerve issues from the lateral part of the pons.

The *ependyma* upon the floor of the fourth ventricle appears to be of abnormal thickness, and the cells of cylindrical epithelium

are large. The ependyma is thrown into folds along the floor, so as to give a crenated appearance to all the upper margins of the sections. It is also to be noticed that a distinctly marked post-*fissure* exists throughout the pons, which is abnormal. Numerous vacuoles are seen throughout the pons, and the blood-vessels are very numerous. When the crura cerebri are reached (fig. 8), the abnormal specimen is seen to differ widely from a normal one, owing to the facts that the roof of the aqueduct of Sylvius is wanting, and that *there are no corpora quadrigemina.* The gray matter lying beneath the floor of the fourth ventricle becomes very thick, and there appear groups of gray nuclei in the external dorsad portion of the specimens, to which nothing analogous is found in the normal specimen. The nuclei of the fourth and third nerves appear to be crowded inward toward the median line and to be displaced downward (ventrad), so that many of the cells lie ventrad of the posterior longitudinal bundle. The number of cells from which third nerve fibres arise, and the number of the third nerve fibres seem to be greater than normal. The third nerve issues from between the red nuclei. The fourth nerve fibres pass outward along the upper border of the red nuclei. The red nucleus is visible, and the decussation of the superior peduncle of the cerebellum, whose fibres are destined to end in this nucleus, is evident in the specimens just below this level. Before the brain was cut it was noticed that the superior peduncles of the cerebellum met and disappeared beneath the lemniscus at a more obtuse angle than normal, and this is evident from the sections, in which the superior peduncles in their full extent were seen to appear suddenly and to decussate at a very obtuse angle.

Many of the sections through the crura cerebri were imperfect. This portion of the brain in the specimen consisted almost entirely of gray matter, and was very difficult to handle, and badly hardened.

As already stated, the peduncles of the crura were absent, the tegmentum alone being present. This left the substantia nigra and red nucleus lying free upon the ventrad surface, with a deep fissure between the two halves of the tegmentum. Some of the gray substance thus lying free crumbled away when the specimen was handled, although great care was taken to prevent such crumbling. It is possible that the portion thus destroyed may have formed a substantia nigra, but in the section as drawn, no such collection of cells (which at this age would not be pig-

mented) was found. The *red nucleus*, however, is intact in several sections and presents about the normal size, shape, and appearance. It is limited ventrad, fig. 8, by a few fibres which pass around it, and which may be likened to the arciform fibres of the medulla, but from their direction cannot be considered as belonging to the superior peduncle of the cerebellum. In fig. 8, the fibres of the third nerve are seen to pass through the red nucleus. The extent and contour of the red nucleus, are well seen in fig. 8, which is made about at its centre. At a higher level a collection of cells of oval shape was seen lying between the red nucleus and the posterior longitudinal bundle. Its shape and appearance resembled that of the corpus Luysii, but its position was not that of that body, which is not found in the specimen. The absence of corpora quadrigemina is evident, but the thick layer of gray substance forming the dorsal border of the crus with its collection of gray cells does not resemble in respect of shape or microscopic appearance, in any degree, these deficient bodies.

It was impossible to obtain sections through the portions of the cerebral mass lying above this level, on account of the imperfect hardening of the specimen. Inspection of the cut surface with a lens showed no appearance of white fibres. There was simply a mass of gray substance which crumbled on the knife, surrounded by a layer of thick connective tissue. Optic tracts could not be found.

To sum up the results derived from the examination of this specimen it may be stated that the chief characteristics are (1) the total absence of pyramidal tracts in all parts of the nervous system, and the deficiency of a portion (about half) of the lemniscus, in the crura, pons, and medulla; (2) the presence of a portion of the lemniscus, and of the interolivary tract; (3) the presence of the formatio reticularis in its entire extent; (4) the presence of the gray nuclei on the floor of the fourth ventricle, with their nerve-roots and nerves; (5) the presence of the peduncles of the cerebellum, and of a normal cerebellum.

These facts are to be brought into relation with the absence in the cord of those tracts which are known to be motor, and the presence of those tracts which are known

to be sensory. It seems evident, therefore, that in this microcephalic brain all the motor tracts in the crura, pons, and medulla are wanting, and that all the sensory tracts are present. The specimen is, therefore, of great value in tracing the sensory tracts.

The sensory tracts in the cord were normal and led to the portions of the medulla which have been stated as sensory. These are the nuclei gracilis and cuneatus, formatio reticularis, and direct cerebellar tract. From the nuclei gracilis and cuneatus some fibres passed through the sensory decussation into the interolivary tract, whence they went upward, formed the outer portion of the lemniscus, and turning upward, passed external to the red nucleus toward the internal capsule. From the nuclei gracilis and cuneatus other fibres passed to the formatio reticularis, there joining fibres which came from the gray matter of the cord; and these together passed up through the medulla, pons, and crura without decussating. The direct cerebellar tract went to the vermiform lobe of the cerebellum—to end in its cortex.

The result of the examination of this specimen supports the conclusion of Flechsig, that tracts develop in the direction in which they convey impulses. Flechsig reports a case [1] which, in some respects, resembles this one. In his case a division had occurred between the brain above and the pons below the corpora quadrigemina; the pyramidal tracts in the pons are wholly wanting, and the lemniscus was diminished to one third of its natural size,[2] while no changes were found in the dorsal half of the pons. In the medulla, the olivary lobes were small, the interolivary tract was reduced to one half its normal extent, the place of the absent pyramids was taken up partly by arciform fibres and partly by "gelatinous substance." The upper decussation was normal. The lower decussation was absent and the fibres of the postero-lateral columns (*i. e.* pyramidenbahn) of the cord were absent.

[1] "Leitungsbahn im Gehirn u. Ruckenm.," p. 120.

[2] In the text the statement is that the lemniscus was reduced to two thirds of its normal size, but in the "Plan d. Mensch. Gehirns," p. 26, this is corrected so as to read one third, as stated above.

The sensory columns of the cord were normal in all respects. (See Taf. xvii., 1–12.)

In Rohon's case [1] there had developed a cephalic mass above the pons about as large as the cerebellum ; and from this a very few fibres issued, forming a thin crusta on each side, but ending in the substantia nigra. In the pons and medulla the same deficiency of longitudinal fibres and pyramidal tracts was found, as in Flechsig's case. But Rohon found a lower pyramidal decussation present. (The plate which he gives as a proof of this pyramidal decussation is a plate of the lower part of the sensory rather than of the motor decussation, as is seen by the fact that the nuclei gracilis and cuneatus can be seen in it, and the number of fibres decussating is much less than in a normal brain at this level.) He, therefore, concludes that in the process of development the pyramidal fibres develop from the brain downward to the point of decussation, and from the cord upward to the point of decussation, and these two parts developing independently unite to form a continuous tract. This conclusion is in direct opposition to the conclusion reached by Flechsig, that the pyramidal tracts develop from above downward along their entire extent.

Up to this time no case has been offered to decide the controversy. But the case here described is such a case, and it proves that the assertion of Flechsig was well founded. In this case the pyramids are wanting in pons and medulla, the pyramidal decussation is wanting, and the continuation of the pyramidal tracts in the spinal cord in its postero-lateral columns is absent. If the pyramidal tracts develop from above downward the case is easily explained. The hemispheres were absent, and the pyramidal tracts failed to develop. If the pyramidal tracts develop partly from below upward, why were they absent in this case, when all other parts of the cord which are known to develop from below upward were present?

While it is impossible to dispute Rohon's assertion that the pyramidal decussation was present in his case, the diagrams which he offers fail to prove it ; and the case of Flechsig, taken in connection with the case here described, prove that Rohon's generalization is erroneous. *The pyramidal tracts develop from above downward in the direction in which they transmit impulses.*

In Rohon's case the lemniscus and interolivary tract are not of normal size, but are more fully developed than in Flechsig's

[1] Rohon: "Untersuchungen über den Bau eines microcephalen Hirnes," Wien, 1879.

case, or in my case. Rohon was able to trace lemniscus fibres to both corpora quadrigemina, which was impossible in the other cases. The other parts were normal in all three cases.

It may therefore be stated that in the brains of three microcephalic infants all the voluntary motor tracts had failed to develop. This failure had not extended to the gray matter of the floor of the fourth ventricle, to the posterior longitudinal bundle, to the formatio reticularis, to the outer half of the lemniscus, to the olivary bodies and interolivary tract, or to the cerebellar peduncles. In some or all of these parts therefore the sensory tracts must lie. It can be shown from pathological facts, that the sensory tracts do not lie in the gray matter of the fourth ventricle, nor in the posterior longitudinal bundle. The olivary bodies are in functional relation with the cerebellum, for they are anatomically joined to it by the inferior peduncles; atrophy of one cerebellar hemisphere is always associated with atrophy of the opposite olivary body; destruction of an olivary body produces cerebellar incoördination. It can be shown that the sensory tracts do not lie in the olivary bodies or cerebellar peduncles. Hence by exclusion it is evident that the sensory tracts must lie in the formatio reticularis, in the lemniscus, and interolivary tract.

It may be added here in order to complete the record of this case, that sections through the superior vermiform lobe of the cerebellum and through the cerebellar cortex showed their structure and nuclei to be normal.

(4) The method of tracing tracts by means of observing secondary degenerations in the medulla and pons has been of more use in determining the motor paths than the sensory. After lesions in the motor area of the brain, either in the cortex or in the internal capsule, the longitudinal fibres lying in the middle two quarters of the crus cerebri and in the anterior (ventral) half of the pons and medulla degenerate

downward.[1] After lesions in the crus or pons involving the parts lying posterior to these tracts a secondary degeneration downward is observed in the inner two thirds of the lemniscus, which can be followed through the interolivary tract and into the olivary body of the same side[2]; and a secondary degeneration upward is observed in the outer third of the lemniscus, which can be followed upward nearly to the optic thalamus, and into the internal capsule of the same side. This latter is the only tract in the pons in which an ascending degeneration has been traced, and its discovery confirms the assertions already stated of various anatomists, that in the lemniscus at least a portion of the sensory impulses pass upward to the brain.

(5) In the absence of further information to be derived from the methods of investigation already considered, it is necessary to consider the more carefully the facts afforded by a study of pathological cases. Lesions of limited extent in the medulla, pons, and crus are not infrequent, but cases available for the present purpose are very rare. This is due to several causes. A lesion of any considerable extent in these parts, especially if situated in the posterior (dorsal) half of the medulla or pons, usually causes sudden death by injuring the centres of the pneumogastric nerves. A lesion in the anterior half of the medulla or pons involves the motor fibres only, and gives no information regarding the exact course of the sensory tracts. The arterial supply of the anterior part of the medulla and pons is derived from the basilar artery, and disease of this vessel may give rise to symptoms chiefly referable to the tracts in the anterior half of the pons. It is from disease of this vessel that lesions of the pons are usually due, softening from

[1] Türck: "Wien. akad. Sitz. Bericht." *Math-nat. Cl.*, 1851, Bd. vi., S., 288. Charcot: "Localization des maladies cerebrales." Paris, 1881.
Brissaud: "Recherches sur la contracture permanente des Hemiplegiques." Paris, 1880.

[2] And in one case as far downward as through the sensory decussation to the nucleus gracilis. Spitzka: *Amer. Jour. of Neurology*, Feb., 1884.

embolism or thrombosis, and hemorrhage being the forms of lesion most frequent in the pons. For these reasons cases of disturbance of the sensory tracts from lesions in these parts are rare. From a lack of accurate knowledge of the microscopic anatomy of the parts many descriptions of lesions are so indefinite as to be of little use in determining the connection of symptoms with lesions. And lastly, the record of the symptoms in many cases is imperfect. Under these circumstances it is not surprising to find that Nothnagel, whose study of local brain lesions is the most complete and careful of any hitherto published,[1] concludes that the cases recorded up to the time of his writing (1879) were not sufficient in number, nor in such accord, as to warrant any statement more definite than the following: "The lesions of the pons producing anæsthesia are usually situated in the lateral portions in the vicinity of the floor of the fourth ventricle, and all kinds of sensations are equally affected." Since the publication of his work, however, a number of cases have been published in various countries and in different journals which, when collected and compared, afford valuable information on this subject. It is to the study of these cases that we now proceed.

It is well known that lesions limited to the anterior (ventrad) half of the pons, and affecting only the transverse and longitudinal bundles, do not produce sensory symptoms. Many cases are recorded which establish this fact. They need not be cited here.[2] It is admitted that disease which does not extend to or involve the parts lying dorsad of the deep transverse fibres of the pons does not give rise to disturbance of sensation. It follows that the sensory tracts

[1] Nothnagel: "Topische Diagnostik der Gehirnkrankheiten," Berlin, 1879.

[2] The following cases are referred to, however, as they illustrate this position: S. M. Burnett, Knapp's *Arch. of Ophthal.*, vi., 469; Janeway, *N. Y. Med. Jour.*, xxxi., 66; Crandall, *Phil. Times*, ix., 313; Pousson, *Progrès Médical*, x., 560; Ballet, *Progrès Médical*, viii., 657; Gautier, *Gaz. Hebdom.*, 1881, p. 701; *Brain*, vii. and viii.; Wernicke, iii., 415; Judell, *Berlin. klin. Wochen.*, 1872, No. 24.

do not lie in the pyramidal tracts, or in the transverse fibres, or in the gray nuclei of the pons lying between these parts—*i. e.*, in the parts ventrad of the lemniscus.

Lesions situated in and limited to the gray matter of the floor of the fourth ventricle, not destroying or compressing subjacent parts, have been recently studied by Weichselbaum,[1] De Jonge,[2] and Luys.[3] Twenty-three cases of such lesions have been collected by these authors. The symptoms in all these cases were referable to destruction of the cranial nerve nuclei. In none of them were there disturbances of sensation in the body. It follows that the sensory tracts do not lie in the gray matter of the floor of the fourth ventricle, a conclusion which is confirmed by the study of cases of bulbar paralysis, in which sensory symptoms do not occur unless the lesion extends to the formatio reticularis.

By exclusion therefore the conclusion is reached that the sensory tracts in the pons must lie between the deep transverse fibres and the gray matter of the fourth ventricle; that is, in the lemniscus or formatio reticularis.

In the following cases these tracts were involved in the pons or in the medulla, and disturbances of sensation were produced. In some of them both the parts mentioned were affected, and then all kinds of sensation were disturbed. In others but one of these parts was affected, and then some kinds of sensation escaped. The study of these cases therefore will establish not only the course of the sensory tracts already indicated by the anatomical and embryological investigations already described, but may indicate the course of different sensory tracts. The cases are first cited and then analyzed.

CASE 1.—Softening in the pons and medulla—sensory symptoms.

[1] Weichselbaum: *Wien. med. Wochensch.*, 1881, No. 32.
[2] De Jonge: *Arch. f. Psych.*, xiii., p. 666.
[3] Luys: *L'Encephale*, 1883, No. 3.

Male, aged fifty, after having suffered for some time from vertigo, was suddenly seized with a feeling of fulness in the head, and a peculiar paræsthesia of the right half of the body, not including the face. This paræsthesia became less after a few days, but there remained a diminution of sensation and a marked ataxia in the right limbs. Eight months after the attack the patient was examined by Dr. Kahler, who found that in both right extremities there was a very marked ataxia upon any motion, which was not increased when the patient's eyes were closed, a loss of the power of perceiving the location of these limbs, and a loss of the sensation of pressure. He had a constant feeling in the right half of the body as if the muscles were contracted. There was no loss of muscular power, and no diminution of tactile sensibility. Other symptoms were nystagmus, paralysis of the left abducens, paralysis of the muscles which open the glottis on the right side. The patient was under observation for two years, during which time there was no change in the symptoms. Cause of death not stated.

Autopsy.—A brown discoloration was found upon the floor of the fourth ventricle in the caudad part of the pons, which was found to correspond to a focus of softening. This focus began in the middle of the left olivary body, and extended upward for 8 mm. through the dorsal segment of the pons in its centre, on both sides of the raphé, but more especially on the left side. The left olivary body and the left interolivary tract were the parts chiefly affected by the lesion. The pyramidal tracts in the pons and medulla, and the lateral parts of the pons and medulla presented a normal appearance.—O. Kahler: *Prager medisch. Wochensch.*, 1879, Nos. 2, 3, and 4.

In the discussion of this case Kahler cites two cases of Leyden's. One of these cases he quotes as follows:

"In a case of Leyden's, in which very marked ataxia of all four extremities was present, the autopsy showed the presence of three small foci of embolic softening lying in the middle of the substance of the pons, but not affecting the pyramidal tracts."[1]

The other case is as follows:

CASE 2.—Softening of interolivary tracts in the medulla—general ataxia.

Male, aged sixty-two, was suddenly seized with vertigo and headache, but was able to walk home. From the onset of the

[1] This case of Leyden's is recorded in his "Klinik d. Ruckenmark—krankheiten," I.

attack he was unable to swallow, and three days afterward was brought to the hospital. He complained of headache and vertigo, of inability to swallow, and of inability to stand or walk, or to use his arms and hands on account of loss of power of co-ordination. Examination showed pupils equal ; no paralysis or anæsthesia of the face ; speech indistinct, especially in pronouncing *T ;* tongue protruded straight, but slowly, and tremor was marked ; total inability to swallow ; constant hiccough ; marked ataxia of both hands and both legs, so that he cannot feed himself, or stand, and walking is impossible, even with assistance. Sensibility seemed perfectly normal in all parts of the body, as was also the sense of pain. In the course of a few days an intense redness of the face was noticed. The symptoms remained stationary. He grew weaker, and became delirious, respiration became irregular, and in six days after the attack he died.

Autopsy.—All parts of the nervous system were normal excepting the medulla oblongata. In the medulla an area of softening was found extending vertically from the middle of the olivary bodies cephalad ½ cm. to their upper limit, and occupying the entire interolivary tract on both sides from the gray matter of the floor of the fourth ventricle to the pyramidal tracts, which latter tracts were slightly involved in the degeneration. The entire raphé was destroyed. The lesion did not reach the pons. The nerve fibres were swollen or atrophied or in a state of degeneration. The process was an acute myelomalacia.—Leyden : *Arch. f. Psych.* vii., pp. 57–61.

CASE 3.—Softening of one lateral portion of the medulla—sensory symptoms.

Male, æt. fifty-six, on waking in the morning found that he was ill, was dizzy, and could not walk, having a tendency to fall to the left, but no paralysis. His left face felt cold, he could not talk plainly, and had difficulty in swallowing. When examined five days after it was found that he could walk only when assisted, and tended to fall to the left, though when seated all motions were good, without ataxia—the right arm trembling, however, slightly. No facial or hypoglossal paralysis, and ocular muscles normal ; swallowing difficult. Sensation was lost in the left side of the face and in the right half of the body and right limbs. The right limbs soon became livid. The patient always knew the position of his limbs. He died fourteen days after the attack, the pulse having been rapid, 130, from the first.

Autopsy.—An area of softening, due to thrombosis of the left vertebral artery, was found in the lateral dorsad caudad portion of the left half of the medulla. The left corpus restiforme, and adjacent part of the floor of the fourth ventricle and formatio reticularis were yellow and softened for 1 cm. in length. The area extended from the viii. to that of the xii. nerve, and involved the restiform body, the adjacent nucleus cuneatus, the formatio reticularis, the ascending v. root, the motor nucleus of the vagus and its fibres. The olivary body, the sensory nucleus of the vagus, and the hypoglossal were not involved.—H. Senator: *Arch f. Psych.* xi., p. 713.

CASE 4.—Softening of one lateral portion of the pons—sensory symptoms.

Male, æt. 50, after suffering for several months from headache and vertigo, was suddenly seized with a sensation as if the entire right side of the body was swollen. When this passed off there was found to be a diminution of the tactile and muscular senses in the right side of the body (not including the face), diplopia, rotary nystagmus, and strabismus due to paralysis of the left sixth nerve, and marked ataxia in the right limbs. The senses of pain and temperature were not affected. Sense of location and of pressure much disturbed. A constant sensation of formication and distension was present in the entire right half except the face. The voice was loud and hoarse, and the patient could not whisper on account of paralysis of the right vocal cord. In the course of the following year a right-sided facial paralysis developed gradually, including the uvula. The disturbance of sensation increased, involving to some degree the senses of pain and temperature and the electro-cutaneous sensibility. The ataxia persisted till death, but true paralysis was not present. The speech became unintelligible as the facial paralysis increased, and the tongue became partly paralyzed. Before death the tongue could hardly be protruded and trembled constantly. He died three years after the onset of the symptoms.

Autopsy.—On the floor of the fourth ventricle beginning near the cephalad boundary and extending back to the striæ acusticæ was found an area of yellow-brown softening. On cutting the pons this was found to lie entirely in its dorsal third, and to the left of the raphé, and to extend from a point five mm. below the corp. quad. down to the upper limit of the olivary body in the medulla. The microscopic examination showed that the primary lesion was a softening due to thrombosis of the pons arteries, in the dorsad

division of the pons, near the raphé, and involved chiefly the formatio reticularis, the lemniscus, and the fibres of the left vi. The following parts were not involved : the pyramidal fibres, the entire transverse fibres of the pons, the middle peduncle of the cerebellum ; the entire nerve nuclei on the floor of the fourth ventricle, the right vi. fibres, and both vii. fibres. A secondary degeneration had developed downward, involving the interolivary tract and the left olivary body in its entire extent.—Kahler and Pick : *Vierteljahrsch. f. d. Prak. Heilk.*, 1879, Bd. 142, S. 96.

Case 5.—Hemorrhage in one lateral portion of the pons—sensory symptoms.

Male, fifty-eight, was suddenly seized with paresis of the left side of the body accompanied by total anæsthesia and loss of the skin reflexes, with total paralysis of the right facial, and abducens, and conjugate paralysis of the left internal rectus, with hyperæsthesia of the left half of the face. The tongue was paralyzed on the left side. Soon after the attack the temperature was lower on the left side of the body.

After one week the paresis had entirely passed away from the left side, but the anæsthesia remained, and was accompanied in the left hand by a loss of muscular sense which gave rise to marked ataxia. The left patellar tendon reflex was increased. The patient's chief complaint was of vertigo and tinnitus aurium. After five months the patient was examined again. There was then right facial paralysis with reaction of degeneration. There was conjugate deviation of the eyes to the left. There was no true paralysis of the extremities, but the ataxia made the left hand and arm useless, and it was slightly atrophied, but there was no contracture. There was partial anæsthesia of the entire left half of the body, greater in the arm than in the leg, and less in the face than in the body ; the sensations for temperature, pain, and pressure being entirely unperceived in the arm. The hyperæsthesia of the right face was no longer present, but the cornea and conjunctiva of the right eye were anæsthetic. Atrophy of the right half of the face, and difficulty of deglutition occurred during the last two months of life. All the symptoms persisted until death, which occurred nine months after the attack.

Autopsy.—Atheroma and miliary aneurisms were present in the larger brain arteries. A clot was found in the gray matter on the floor of the fourth ventricle, on the right side, in its upper half. The lesion involved chiefly the formatio reticularis, and the lemniscus. It had destroyed the right vi. and vii. nuclei and had

involved the post longitudinal bundle, thus probably producing the left internal rectus paralysis. It had reached and involved slightly the ascending root from the nucleus of the right v. and had also destroyed the descending root of the v. on the right side (which is thought by Meynert to go to the left v. nucleus). It had not reached the viii. centres. It did not affect the pyramids. Secondary degeneration was found to have taken place from the clot downward in the lemniscus, and this was traced to the inter-olivary zone and into the olivary body of the *same* side, this being much atrophied.—Meyer: *Archiv. für Psychiatrie*, xiii., p. 63.

CASE 6.—Hemorrhage in one lateral portion of the pons; sensory symptoms.

Male, æt. 41, had an attack of vertigo followed by difficulty of motion in the right arm and leg with formication in them and in the left half of the face, and diplopia. The disturbance of sensasation remained for three months, while that of motion disappeared in a few hours. Nearly two years after the attack a second one, precisely similar, occurred, and he then entered Senator's division of the Augusta hospital. Examination showed analgesia in the left second branch of the trigeminus; neither eye could be turned to the left; paresis of the right arm and leg, which soon became almost total. Soon after paresis of the left facial and right hypoglossal developed and swallowing became difficult. The patient had a great diminution of the power of sensation of touch, pain, temperature, and the position of the paralyzed limbs, and the skin reflexes were here diminished, while the right patellar reflex was increased. These symptoms all increased in intensity, and it was also noticed that the right hand and forearm were warmer, damper from sweat, and more livid than the left hand and forearm. There were no urinary symptoms. His intelligence was undisturbed, and the special senses were normal. The anæsthesia and paralysis of the left half of the face and right limbs were nearly complete at the time of death, and the eyes were both directed to the right constantly—the pupils being normal. Seven weeks after his second attack he died.

Autopsy.—Atheroma and thrombosis of the left vertebral and posterior half of the basilar artery with softening of the pons and medulla were discovered. A hemorrhage was found on the floor of the fourth ventricle 1 mm. wide, 1 cm. long in the median line in the upper half. A second clot lay over the sixth nucleus. Section showed an extensive area of softening in the left half of the pons and medulla, involving chiefly its dorsad part near the gray matter

of the fourth ventricle, which appeared to be sunken in. This extended from the nucleus of the vi. to that of the xii. nerve, and to the lower end of the olivary body, being narrower at the ends than in the middle, and lying diagonally to the long axis of the pons, so that above, it was nearer the raphé and the dorsal surface than it was below. It therefore destroyed the formatio reticularis, all the nuclei in part from the vi. down to and including the hypoglossal, the lemniscus, and interolivary tract, the median part of the olivary body, the deepest fibres of the pyramidal tract, the ascending trigeminal root and the solitary bundle, and the median part of the restiform body. It touched the direct cerebellar column at the lowest limit. The right half of the pons and medulla were normal.

Remarks.—As the vi. nucleus was not involved it is necessary to suppose a centre for the conjugate movements of the eyes, lying outside of the vi. nucleus, which was destroyed by the lesion. The fibres to the right xii. were destroyed, while a portion of the left xii. nucleus remained, hence the deviation of the tongue to the right on voluntary motion.—H. Senator: *Arch. f. Psych.*, xiv., p. 2.

CASE 7.—Hemorrhage into one lateral part of pons; sensory symptoms.

Male, æt. fifty-eight, was suddenly seized with faintness and vertigo, and then noticed that his right arm and leg were numb as if asleep, but were not paralyzed. The numbness continued for some months, during which time his vision was blurred. It then passed off, but he continued to use his hand in a clumsy way. Two years after the first attack he had an aggravation of his symptoms—the numbness increasing, and his tongue becoming paralyzed for a few hours. He was then examined by Dr. Spitzka, who found that the man's movements with his right leg and arm were jerky and clumsy, and that he felt insecure in walking in the dark. There was ataxia of the right arm, knee-tendon reflex, exaggerated on right side. Skin reflexes absent on right side. No trophic disturbances. Tongue deviates to right. Speech thick after talking some time. Tremor of lips is present. Electric reactions normal. Tactile sensibility impaired in right hand, and on dorsum of forearm; also in right foot. Sensation of pain is quite acute. Sensation of temperature quite impaired. He cannot judge differences of weight five times as great as those recognized on the left side, and cannot judge of the nature of surfaces with his right hand. The direction in which a cold rod is laid

upon his skin is not accurately judged on his right side in arm and leg, and on the body to a less degree—to within three inches of the median line. Marked loss of muscular sense in arm and leg. Sensation slightly impaired on right cheek and lips. After a year the numbness extended to the left foot, and three months later the symptoms had increased in intensity to a marked degree, and he began to have difficulty in swallowing. Six months later the trouble in speech had increased, and he stumbled in speaking. At this time he had two attacks of dizziness and fainting, and, in addition to former symptoms, contraction of right pupil was noticed. Four months after this he became somnolent and apathetic, yawned a great deal, breathed irregularly, and œdema of the right hand, with a decided right hemiparesis and paraplegic weakness of the lower extremities was present. He did not appreciate when his bladder was full. At this time he took to his bed. Soon after he noticed tingling of *both* lower, and a subjective sense of stiffness in both upper, extremities, more marked on the right side. One month later left ptosis developed, and the tongue now protruded to the left. A few days after this involuntary discharges began, his intellect for the first time began to wander, and he suffered much from a feeling of coldness, though the room was so hot that he was in a profuse perspiration, and his temperature was normal. Six days before his death his breathing became stertorous, and right ptosis with extreme myosis was found, the previous left ptosis having passed off. He died comatose at age of sixty-two.

Autopsy.—Dura normal. Opacities in the great falx. Cerebospinal fluid increased in quantity. Vessels calcified. In both hemispheres numerous capillary hemorrhages and small spots of softening were found, none more than 1 cm. in size. In the cerebellum several perivascular hemorrhages were found in the dentale nucleus and two miliary patches of softening, which were found to be recent, as were all the lesions except the one in the pons. In the pons an old hemorrhagic focus was found of irregular shape, lying wholly in the *left* half, and occupying the area of the lemniscus, being thus ventrad of the formatio reticularis, and dorsad of the longitudinal fibres of the pyramids.

The focus of disease consisted of a cavity with partly organized walls and intense contiguous tissue-changes. At the level of the motor nucleus of the v. the cavity was merely a slit, while the area of tissue-change around it occupied nearly the entire field of the lemniscus, failing to reach the raphé or the motor root of the

v. on either side. In lower sections the cavity became larger, and advanced ventrad involving the transverse fibres of the pons below the lemniscus. The contiguous tissue-change involved the raphé, and extended across the median line 2½ mm. The cavity extended caudad as far as to the facial-nerve nucleus, which, however, was not involved.

An area of *descending degeneration* was found, involving the lemniscus and the interolivary tract on the left side down to the sensory decussation, where it was followed through the decussation into the opposite side of the medulla, where it involved the nucleus gracilis to a considerable extent, and the nucleus cuneatus in a lesser degree. The olivary body on the side of the lesion was not involved in this degeneration, although the field around it on all sides was involved. The internal accessory olivary nucleus was not involved. An ascending degeneration was traced, involving the middle third of the lemniscus as high as the level of the corp. quadrigeminum post. It could not be traced higher, but it was impossible to identify that division of the lemniscus which passes into the thalamic region of the left side.—E. C. Spitzka: *Amer. Jour. of Neurology and Psychiatrie*, Nov., 1883. Published Feb., 1884.

Spitzka cites the following case of descending degeneration in connection with his case:

CASE 8.—Among a number of cases of secondary degeneration in the pons medulla and cord studied by Homén (*Virchow's Archiv f. path. Anat.*, Bd. 88, S. 61–84), one is recorded in which a focus of softening in the left half of the pons was followed by degeneration downward, both of the pyramidal column and of the lemniscus. The latter was destroyed by the focus of disease at the level of the common nucleus of the abducens and facial nerves. Below this point the secondary degeneration of the lemniscus was traced as far as to the sensory decussation, lying in the lemniscus and interolivary tract. The case is accompanied by a very meagre history, which merely states that the patient was hemiplegic on the right side for three years. No reference is made to any sensory disturbance. It is, of course, impossible to draw any physiological conclusions from this case, but inasmuch as it supports the view of Flechsig that a portion at least of the lemniscus degenerates downward, it is cited here.

CASE 9.—Tubercle of the pons; anæsthesia of the face alone. Male, æt. fifty-eight. Began to suffer in July from headache,

diplopia, and difficulty in opening the mouth. In August an examination showed the presence of left facial paralysis including all the branches of the nerve ; spasm of the left masseter ; no affection of the tongue ; ptosis of both eyes, especially of the left ; conjugate deviation of both eyes to the right, it being impossible to turn the right eye beyond the median line, or to move the left eye toward it. Pupils contracted. Sight impaired by old cataract. Smell, taste, and hearing normal ; possibly a slight degree of deafness in the left ear. Loss of sensation to all stimuli and numbness of the right side of the face and neck. No paralysis, ataxia, or anæsthesia in the body or limbs. No urinary symptoms. These symptoms, with headache, vertigo, and vomiting, persisted till death occurred in October.

Autopsy.—On the floor of the fourth ventricle in the middle of its left half a tumor 2 cm. wide and $1\frac{1}{2}$ cm. long was found, which had involved the lateral-dorsad part of the pons, but did not reach the transverse fibres. The tumor was a tubercle, and was not surrounded by any zone of softening. It involved the left common abducens-facialis nucleus, the left facial nucleus and genu, the motor root of the trigeminus, and the descending root of the trigeminus on the left side, which decussates, according to Meynert. At a lower level it involved the eighth and ninth centres on the left side. The lemniscus and the greater part of the formatio reticularis were unaffected.—Wernicke : *Arch. für Psychiatrie*, vii., p. 513.

CASE 10.—Hemorrhage in one half of pons ; sensory symptoms.

Female, æt. forty. In June, 1881, she had a sudden attack of vertigo and loss of consciousness, which had been preceded by a continuous occipital headache for several days. On recovering consciousness she found the right half of her body totally paralyzed, and noticed that the left ear was deaf. She did not notice any loss of sensation. A few days after the sight became dim in the left eye, the cornea became inflamed and cloudy, and, finally, she lost the sight entirely in that eye. Some weeks after the attack her right limbs became rigid and remained so three months. She never had any embarrassment of respiration. In October she was seen by Dr. Miles. At that time the right leg could be moved slighly, and the right arm fairly well, but its motions were ataxic, and power of grasp was much less than in the left hand. There was bilateral facial paralysis involving all the branches of both nerves, and producing immobility of the face and defective

pronunciation of the labials. The tongue was protruded to the right with an irregular, uncertain motion. There was no muscular atrophy, and faradic reaction was normal in all the muscles. For five months after the attack there had been a tonic contraction of the muscles of mastication, so that the teeth were not separable more than a quarter of an inch. There was no trouble in swallowing. The nails were found to be growing faster on the paralyzed side, but were not ridged. There was incontinence of urine and fæces. Tactile sensibility was normal, except over a small area of the lower right face, and entire left half of the forehead, and the middle third of the flexor surface of the right forearm, which areas were also analgesic. Tactile sense of the tongue was good. Taste and smell intact. Total deafness of the left ear; right ear normal. Speech was slow, jerky, and drawling, but there was no aphasia. There was no paralysis of the ocular muscles, and no ptosis. The left cornea was opaque from the presence of extensive pannus, and much hypertrophied.

In December, 1881, the spasm of the muscles of the left side of the jaw relaxed, and the right facial paralysis almost disappeared. By February, 1882, the left eye had atrophied and the cornea had sunk in. At times flashes of heat and redness, with tingling, occurred in the right half of the body and limbs, and the entire side was redder than the left. There was no polyuria. Patient was emotional, laughing and crying easily, but otherwise there was no loss of intellectual power. She grew weaker, bedsores developed, and she died May 21, 1882.

Autopsy (twenty-eight hours p. m.).—Dura normal. Pia opaque and thickened, but not adherent. Arteries atheromatous. On the basilar artery at the inferior margin of the pons, a fusiform aneurism was found of the size of a bean. The left vii. was seen to be smaller than the right vii. The hemispheres were slightly atrophied. The lateral ventricles were distended with serum, and the ependyma thickened. The brain was wet but normal. On the floor of the fourth ventricle, in its left half, just above and external to the eminentia teres, a small yellow irregular depression was seen. Transverse section at this point showed an old hemorrhagic focus of stellate form, extending from five mm. below the caudad border of the pons, five mm. internally to the floor of the ventricle cephalad, nearly to the superior border of the pons, growing smaller as it ascended. The centre of the lesion lay anterior (ventrad) to the genu nervi facialis of the left side. From this point two arms extended forward, nearly reaching the

superficial transverse fibres of the pons. Nowhere did it approach the median line. In the right half of the pons at a point slightly anterior (ventrad) to the vii. nucleus was another focus, also old, of the size of a No. 5 bird-shot. Microscopic examination showed the presence of miliary aneurisms, also a descending degeneration of the crossed pyramidal tract in the right half of the cord, and of the direct tract in the left half. The left v., vii., and viii. n. nuclei were involved.—F. A. Miles: *Archives of Medicine*, Aug., 1882.

The lesion involved both the formatio reticularis and the lemniscus in the pons, as can be seen in the drawing accompanying the case.

CASE 11.—Hemorrhage in one lateral portion of the pons; sensory symptoms.

Female, æt. forty-four, was suddenly seized with giddiness and faintness, but did not lose consciousness. Gradual loss of power supervened in the left arm and leg, with loss of speech and dimness of vision. The next day, on admission to King's College Hospital, there were found paralysis of the right facial, spasm of the left orbicularis palpebrarum, paralysis of the left hypoglossus, and of the left arm and leg, most marked in the extensor muscles; anæsthesia (partial) and analgesia of the left arm and leg, but no anæsthesia of the face. The symptoms increased during the following two weeks, and paralysis of the right abducens and difficulty of deglutition developed. During the last six days there was constant twitching of the right extremities, and the bladder was paralyzed. She died eighteen days after the attack.

Autopsy.—A fusiform hemorrhage was found in the pons, extending along almost the whole length of the right side. It was 2 cm. long, and its anterior (cephalad) margin was 0.5 cm. caudad of the corp. quad. post., while its caudad margin was 1 cm. cephalad of the pyramid of the medulla. Its widest portion was situated at a depth of $1\frac{1}{2}$ cm. from the ventrad surface of the pons. It did not cross the raphé, but opposite its posterior extremity, in the left half of the pons, was a small clot the size of a hemp-seed, which lay at a depth of 1 cm. from the anterior surface. The clot was wedge-shaped, and its apex projected slightly into the iv. ventricle.—F. Willcocks: from Clinic of Dr. Johnson, King's College Hospital Report in *Brit. Med. Jour.*, 1881, i., p. 272.

CASE 12.—Tumor in one lateral half of pons; sensory symptoms.

Female, twenty-eight, was suddenly seized with an acute pain in the right side of the head, and lost her consciousness. On recovering from the attack, she was found to have total paralysis of the left hand, paresis of the left leg, and anæsthesia in both these limbs; also paralysis of the right motor v., right vii., right xii., and anæsthesia of the right half of the face. She was deaf in the right ear. Her speech was imperfect, and she had difficulty in swallowing. In this condition she lived fourteen months, and died of exhaustion.

Autopsy.—A semi-cartilaginous fibrous tumor was found in the dura and pia upon the right side of the pons and medulla oblongata. It extended from the point of exit of the v. backward for two inches, enclosing the right vertebral artery. The surface of the right crus cerebelli was softened, and so was the pons, upon which the tumor lay. It was incorporated with the substance of the right side of the medulla, and had produced softening throughout its tracts. The left side of the medulla was normal. The roots of all the right cranial nerves, from the v. to the xii., were compressed.—*Amer. Jour. Med. Sc.*, vol. xxviii., p. 106 (1841).

CASE 13.—Softening of pons; sensory symptoms.

Male, æt. seventy-four; a complete left hemiplegia of the arm and leg, with impairment of sensation gradually developed, and remained for one year before death, the right side never being affected. The slighest abrasion on the left side produced marked ulceration. There was no incontinence of urine or fæces. He died suddenly.

Autopsy.—The meninges were congested, and there was some effusion in the ventricles and under the arachnoid. In the falx cerebri were found four small pieces of bone one fourth inch thick. On the floor of the lateral and fourth ventricles granular excrescences resembling boiled sago were found. In the pons was a wedge-shaped area of brown softening. It spread over the anterior surface of the pons in its entire width, and one half an inch antero-posteriorly. Its apex extended on the right side to a depth of three eighths inch, and involved the longitudinal fibres. On the left side it was superficial, and involved only the transverse fibres. —J. B. Tuttle: *Phil. Med. Times*, xii., p. 350.

CASE 14.—Tumor of one half of pons; sensory symptoms.

Female, æt. seventeen; when admitted to the hospital was so stupid that no history could be obtained. On admission the following symptoms were found: complete paralysis and anæsthesia of the left half of the face; complete paralysis and partial anæs-

thesia of the right arm ; paresis and impairment of sensation in the right leg, though she could walk ; loss of smell in left nostril (tested by ammonia, which indicates anæsthesia) ; no paralysis of the tongue ; loss of hearing in the left ear ; total paralysis of the left eyeball, without strabismus; conjunctiva and cornea acutely inflamed ; deglutition difficult, but appetite good ; no vomiting ; pulse rapid and weak ; temp. varied slightly from normal ; involuntary evacuations. Her mental faculties were so blunted that it was impossible to obtain reliable replies. She went into a condition of coma and died. (Duration not stated.)

Autopsy.—The pons was greatly distorted, and enlarged on the left side. Its surface was nodular, and its margins overlapped the medulla and crus. The left crus cerebri was also enlarged, and nodular. The left pyramid was compressed and indented, and the right pyramid pushed aside. The floor of the fourth ventricle was widened and bulged upward on the left side. The tumor was spherical in shape, occupied the left half of the pons, and had pushed the raphé to the right. No microscopic examination. No description of sections.—F. A. Miles : *Arch. of Medicine*, Oct., 1881.

CASE 15.—Tumor of one half of the pons ; sensory symptoms.

Male, æt. eight. Nov. 1, 1877, he suddenly fell down, and on being helped up could not stand, and trembled greatly. Three days subsequently he had a similar attack, but this did not interfere with his going to school all the month. His teacher noticed that he was very clumsy, but did not think him stupid. For three weeks prior to Dec. 12th he suffered from darting pains through his head, occasional vomiting, and weakness in his left hand. When examined Dec. 12th there were found occipital headache, right facial paresis, head inclined to the left, ptosis of left eyelid, paresis of left hand, and an unsteady gait. In a week the symptoms had increased in degree, the left pupil was dilated, but the ptosis had disappeared in the left and appeared in the right eyelid. His skin was cool—pulse 80, regular—appetite good. There was no intellectual disturbance. Dec. 29th.—Vomiting is now associated with the paroxysmal headache, and at the same time his bowels move. His speech is indistinct, and he is now very garrulous, talking constantly. During January his mind became much weakened ; his special senses were not impaired ; strabismus of the right eye appeared, the ptosis remaining ; right half of face became anæsthetic ; he could no longer stand or walk, and his head seemed too heavy for the muscles which support it. The

optic discs were normal. The left hemiplegia became complete, and his right foot was kept in motion constantly; the pulse became irregular and rapid, and on Feb. 10th he died of paralysis of the pneumogastric nerves.

Autopsy.—The right side of the pons was much larger than the left; soft and white, in its entire extent. The change extended along the middle cerebellar peduncle into the right hemisphere of the cerebellum. The fifth nerve could not be traced through the mass. This portion of the pons consisted of round and oval cells with few nuclei in a granular stroma, with many vessels. The tumor was a soft glioma. No sections, and no accurate localization.—J. C. Mackenzie: *Cincinnati Lancet and Clinic*, iv., p. 150.

CASE 16.—Abscess in one half of pons; sensory symptoms.

Male, æt forty-four, while suffering from an abscess of the arm developed suddenly paralysis of the right facial, anæsthesia of the right trigeminus, paralysis of the left hypoglossus, and anæsthesia of the left side of the body. On the next day swallowing and speech became difficult, and spasms of the left arm and leg began and were followed by paresis. Two days after respiration became difficult and he died.

Autopsy.—(By Huguenin.) An abscess was found in the right side of the pons, involving the dorsad half almost in its entire extent without involving either the inferior or superior peduncles of the cerebellum. In the lower part of the pons the abscess had broken through the deep transverse fibres and had reached the longitudinal fibres of the anterior portion.—Bircher, *Schweitzer ärtzbl. Corresp. Bl.*, 1881, No. 4, quoted by Wernicke, *l. c.*, iii., p. 417.

CASE 17.—Tumor of one half of the pons; sensory symptoms.

Male, æt. thirty-two, syphilitic, was kicked in the head by a horse five years before the acute symptoms set in, but ever since that time had suffered from nocturnal headache, and occasional attacks of vertigo. Four weeks before his admission to the hospital he had a fall on the ice and hit his head. No ill effects of this fall manifested themselves until one week after it when he suddenly had an attack of vertigo and fell down. A few days after his sight became dim and he noticed a weakness of the right arm and leg. On admission it was found that his memory was defective so that history was uncertain. He was able to walk but his entire right side was paretic except the upper branch of the facial nerve. It was noticed that the right side of the forehead wrinkled more promptly than the left. Sensation was diminished

in the right limbs and in the left side of the face. Hearing, smell, and taste normal. Both eyes were constantly directed to the right and they could not be turned to the left of the median line. They were fixed and staring; pupils equal, and normal; in accommodation the axes converged slightly; the fundus was pale; the outline of the disc irregular, and the disc hyperæmic and opaque. The patient got worse gradually, and had to stay in bed on account of the vertigo. He had frequent attacks of epistaxis, and became anæmic and weak. On the day before death the limbs of both sides seemed equally paralyzed, and the mouth was drawn to the right side. There was decided loss of sensation in the right limbs and left side of the face. The eyes still deviated to the right and the pupils were small. He died of exhaustion. (Duration of illness not stated.)

Autopsy.—In the left squamous portion of the temporal bone a fracture of the skull—not depressed—was found, and here the dura was adherent, and on its inner surface was a small hard yellow nodule the size of a pea which had caused a slight depression in the left first temporal convolution at the junction of its middle and posterior thirds. A tumor one half inch in diameter was found in the body of the pons, causing a bulging of the floor of the fourth ventricle in its cephalad left part. The tumor was limited to the cephalad left quarter of the pons and did not cross the median line. It had pushed apart the anterior and posterior surfaces of the pons and had not disturbed their integrity(?). Microscopic examination showed it to be a gumma. There was descending neuritis of the optic nerves.—C. K. Mills, JOUR. MENT. AND NERV. DIS., July, 1881.

CASE 18.—Hemorrhage in one half of the pons; sensory symptoms.

Male, æt. twenty-four, suffered from headache for several days, and then from numbness and formication in the left arm. On Oct. 14th, was suddenly paralyzed in the left extremities and left side of the tongue and right side of the face, and articulation was impossible. The left extremities were anæsthetic. The paralysis subsided somewhat in the extremities before his death, which occurred on Nov. 24th.

Autopsy.—A clot was found in the right half of the pons, just beneath the floor of the fourth ventricle. It extended nearly to the corpora quadrigemina. The clot was hard, yellowish, and fibrinous, and the surrounding substance was normal. Very small hemorrhages were also found, one in the centrum ovale and one

in the left corpus striatum.—Mavot: quoted by Wernicke, *Lehrbuch d. Gehirnkr.*, II., p. 95.

CASE 19.—Tumor of one lateral half of pons; sensory symptoms.

Female, æt. forty, after suffering from headache, vertigo, and vomiting, with deafness and diplopia, developed gradually paralysis of the right abducens, of the left facial, of the left arm and leg, and total anæsthesia, with loss of muscular sense in the paralyzed extremities and in the left half of the trunk. Then followed attacks of pain shooting down the left arm and leg. The tongue protruded straight, and speech was perfect. The special senses were normal. Difficulty in chewing, with spasm of the masseters developed before death.

Autopsy.—In the substance of the right half of the pons a tubercular tumor was found, 2 cm. in diameter, which produced a bulging upward of the floor of the fourth ventricle. The tumor began at the cephalad border of the pons, and extended down to the inferior peduncle of the cerebellum, lying chiefly in the dorsad part of the pons near the floor of the fourth ventricle. The right middle peduncle of the cerebellum and the left half of the pons were normal. It was surrounded by a zone of softened tissue, which extended upward in the right crus cerebri nearly to the optic thalamus (ascending degeneration ?). In the lower extremity of the right posterior central convolution upon its surface a second tubercle was found of the size of a pea.—Mavot: *Bull de la Soc. Anat.*, Paris, mars, 1875, quoted by Nothnagel, *l. c.*, p. 121.

CASE 20. Softening of one half of the pons; sensory symptoms.

Male, æt. thirty-three, had for several days a feeling of numbness, cold, and weakness in the right arm and leg, and headache. Then paralysis in these limbs developed suddenly without loss of consciousness. Examination showed paralysis of the entire right side, with anæsthesia, the face being included. Duration of illness not stated.

Autopsy.—A thrombosis of the basilar artery had produced an area of softening in the pons, which involved its entire left cephalad dorsad half. Details of the lesion are wanting.—Nothnagel: *Topische diagnostic d. Gehirnk.*, p 112.

CASE 21.—Sarcoma compressing the pons and crus; ataxia.

Male, æt. nine, began to use his right hand in an awkward manner in May, 1874, and in the course of a few months had lost power almost completely in the right arm. He then began to

suffer from headache, nausea, vomiting, double vision followed by strabismus, due to paralysis of the left abducens He had occasional twitchings in the right hand, but no convulsions. During the next year the paralysis extended to the right leg, and there was a staggering gait. There developed ataxia and rigidity in the fingers of the paralyzed hand. Optic neuritis followed, and he suffered much from pain in the legs. His symptoms increased in severity until death, which occurred in April, 1880.

Autopsy.—A sarcoma was found upon the base of the brain, pressing upon the left crus cerebri and the pons. (Details wanting).—E. C. Seguin : "N. Y. Neurol. Soc. Rep.," JOUR. MENT. AND NERV. DIS., Jan., 1882.

CASE 22.—Sarcoma of one half of the pons ; ataxia and sensory symptoms.

Male, æt. thirty-eight, fell and hit his head on Jan. 13th, and since that time has suffered from headache, vertigo so severe that he fell at times, and occasional attacks of nausea, and pain and weakness in his right shoulder. Was admitted to the hospital Feb. 23d, when a paresis of the right arm with numbness, impairment of speech and of deglutition, and "a distortion of the features, most marked on the right side of the face" (left facial paralysis ?), were found. His gait was reeling. His memory was so defective that the history was obtained with difficulty. He was habitually constipated. There was no change in his condition up to June, when he had two apoplectic seizures, occuring at an interval of two weeks, and died.

Autopsy (twenty-four hours, p. m).—The lateral ventricles were distended with serum. A tumor, of the size of a hickory nut, was found situated in the pons, and involving chiefly the dorsad surface of the left lateral half. A second tumor of the same size was found in the extreme posterior projection of the right lobe of the cerebellum. Microscopic examination showed them to be round-cell sarcomata.—G. Hart : *St. Louis Med. and Surg. Jour.*, vol. xiii., p. 571.

CASE 23.—Glioma of the pons ; ataxia.

Male, æt. six and a half, in Nov., 1873, fell on the back of his head. Two days after this he had a headache, and two weeks after his gait became irregular and ataxic, so that he reeled in walking. He was restless at night, and had headache frequently, but no vomiting or constipation. In Jan., 1874, his speech became indistinct and jerky, and his memory began to fail. In April he had become very stupid, and his speech was slow and

unintelligible though he tried to talk much. There was marked ataxia of the head and all the limbs, but no paralysis or anæsthesia. There was exophthalmos, and all the motions of the eyes were performed slowly, but there was no disturbance of vision. Hearing was good. Later in the month he had involuntary evacuations of urine, and the left pupil was dilated. In May, vomiting, intermittent pulse, dimness of vision, and paresis of the facial muscles began, and an ophthalmoscopic examination showed atrophy of the left disc, and congestion of the right disc. On the 8th he became comatose, and on the 9th he died of apnœa.

Autopsy.—The pons was found to be enlarged in all directions, measuring two inches long, two and a half inches wide, and one and a half inches thick. In a depression along its centre ran the basilar artery, which was much stretched. The tubercula quadrigemina were pushed up and flattened; the cephalad anterior portion of the fourth ventricle was occupied by a rounded swelling, which was firm on the left side and elastic on the right side. The anterior pyramids at their entrance into the pons were elevated, but their point of entrance was deep and normal. Microscopic examination showed the existence of small, round, and polygonal cells, with granular matter in a stroma of thickened neuralgia, throughout the pons. (Glioma.) There was an atrophy of the optic nerves. Lateral ventricles distended by serum. —Gibney: *Amer. Jour. Med. Science*, July, 1875.

CASE 24.—Abscess compressing the pons; sensory symptoms.

Male, æt. twenty-one. In Sept., 1869, began to suffer from headache, and, on the 10th, left facial paralysis developed and remained. He entered Massachusetts Hospital Oct. 27th, when left seventh and twelfth paresis was noticed, and it was found that, in walking, he was dizzy and inclined to fall to the left. Subsequently, paresis of the left arm and leg developed, and, one month after, involuntary motions of the hand and arm occurred, with stiffness on passive extension. One week later, inco-ordination was so marked in his legs that he could not stand. The sensation of the limbs was not affected, but he had pain in the left side of the face and on the inner side of the left arm, and the left cheek and eyeball were anæsthetic. There was a gradual loss of vision, first in the left and then in the right eye. He had been deaf in the left ear for years. There was occasional flushing of the face. During three days in December he lay in a stupor. In January, nausea and vomiting began, the head ache became intense, and, at last, dysphagia developed, and he died Feb. 18, 1870.

Autopsy.—Pia strongly injected at the base; four ounces of serum in lateral ventricles. Brain-substance firm with numerous puncta vasculosa. Left side of the cerebellum was prominent and half as large again as the other side, the medulla and pons being pushed to the right. The left fifth, sixth, seventh, and eighth nerves were covered in, and destroyed by, a morbid growth; the ninth, tenth, and eleventh were stretched over the abscess to be described; twelfth normal. Along the side of the medulla was a swelling—soft and fluctuating, which was found to be an abscess containing green pus; its cavity was as large as a walnut, was lined with a membrane covered with villi. It extended upward and inward under the medulla and lower edge of the pons, not affecting the cerebellar peduncles except by pressure, seeming to lie between the lower and middle peduncles and to press them apart. The lower peduncle was spread out over its surface; it extended down almost to the lower part of the left cerebellar lobe, near the median line, the corpus dentatum not being affected. On the outer side of this abscess was a second one, as large as a hickory nut, separated from the first by a layer of cerebellar tissue, and occupying the upper part of the upper portion of the left cerebellar lobe. The fourth ventricle was larger than normal, being stretched over the abscess. The morbid growth was a glioma consisting of cells in groups and fibrous tissue.—S. G. Webber, *Boston Surg. and Med. Jour.*, vol. lxxxii, p. 289.

CASE 25.—Softening of the pons; hemiplegia, subsequently opposite facial palsy.

Male, suffered for four months from paresis of right arm and leg, with slight weakness of the right side of the face. He was then attacked with severe pain in his left jaw, which radiated from behind and below the left ear, over the side and front of the head and face, and, the next day, the left half of the face was found paralyzed. The pain continued for a month, and was associated with hyperæsthesia of the second branch of the fifth nerve. Reaction of degeneration not present. Two months after this attack, diabetes developed. All the symptoms persisted until nine months from the beginning of his illness, when he died suddenly. No mention is made of any disturbance of sensation, except in the second branch of the fifth nerve—on the left side.

Autopsy.—There was a depression of the convexity of the pons on the left side. Opposite to this, in the middle of the pons, was an irregular area of softening, situated in the pyramidal fibres, opposite and a little below the origin of the fifth nerve. Above, the

softening was in two foci, one of which was a cavity, the other being occupied by granular debris ; but, a little lower, these had blended into one of rather smaller size. The fibres of the fifth and seventh were both apparently undamaged, but the lesion in its lower part was close to the fibres of the seventh. In the middle of the pons, the left ascending sensory nucleus of the fifth presented two small foci of softening. The anterior pyramid of the left side was completely degenerated, and the degeneration was traced through the decussation to the opposite lateral column of the cord.—Gowers : *Brain*, pt. vii, p. 474.

CASE 26.—Tumor of the pons ; no sensory symptoms.

Male, æt. fifty-four, suffered for three months before his death from severe continuous headache, and two months before death noticed that his vision was disturbed by the fact that his eyes were constantly turned to the right. There was no diplopia. On examination, the head and eyes were found to be turned to the right, and the eyes, moved together, could not turn beyond the median line to the left, though the right eye alone could be turned for some distance to the left beyond the median line. Pupils equal and mobile. The position of the face was not due to paralysis or contracture of the muscles of the neck, and he could turn his head in any direction. *No paralysis or loss of sensation.* He had some dizziness, and staggered in walking. He died of pneumonia.

Autopsy.—No meningitis. No lesion in the hemispheres. A tumor was found in the pons at a level one cm. below (caudad) the apparent origin of the v. on the left side. It was so situated in front of (cephalad) the eminentia teres that it involved the course of the fibres of the left abducens, and by a little prolongation across the raphé toward the right side, interrupted the fibres of communication between the vi. and iii. centres. It did not involve the common nucleus of vi. and vii. It interrupted the posterior longitudinal bundle and the adjacent part of the raphé. No other lesion was found. The tumor was the size of a small nut. The position of the head was regarded not as compensatory for the position of the eyes, but as due to a severing of fibres joining the rotatory muscles of the head with their reflex centres. —Quioc : *Lyon Médicale*, 1881, July, Nos. 29, 30.

ANALYSIS OF THE SYMPTOMS.

The large number and varied character of the symptoms present in these cases make an analysis desirable ; and in

order to reach any conclusion from the study of the cases, as to the tracts conveying sensory impulses, such an analysis is necessary.

The Sensory Symptoms.

I. Disturbances of the tactile sense.

The tactile sense was disturbed to some degree in twenty-one cases. In cases 1, 2, 21, 22, 26 it was not affected. The distribution of the disturbance was as follows:

1. Anæsthesia of the right side of the face in cases 5, 7, 9, 10, 12, 15, 16, 20. Anæsthesia of the left side of the face in cases 3, 6, 10, 14, 17, 24, 25. Inflammation of the cornea coincident with the anæsthesia, occurred in cases 5, 10, 14.

In cases 3, 5, 6, 12, 14, 15, 16, 17, 24, 25, the anæsthesia of the face was on the same side as the lesion, and the lesion involved the ascending root of the trigeminal nerve in some part of its course in the medulla or pons, and did not involve the descending root. In cases 7, 9, and 20 the anæsthesia of the face was on the opposite side from the lesion. In cases 7 and 9 the lesion was situated so high in the pons (cephalad) as to involve the descending root of the trigeminus to some extent, but too high in the pons to affect the ascending root at all. The anæsthesia was slight in degree and was limited to the cheek and lip in case 7. It was well marked in case 9. In case 20 all details of the extent of the symptom and of the position of the lesion are wanting. In case 10 both sides of the face were anæsthetic, but the anæsthesia was limited to the upper branch of the trigeminus on the side of the lesion, and to the lower branch of the trigeminus on the side opposite to the lesion. In this case the lesion was so situated as to involve a small portion of the ascending root of the trigeminus and also a portion of the descending root of the trigeminus on the same side.

The conclusions to be reached from these facts are as follows:

(*a*) *Lesions affecting the ascending root of the trigeminus produce anæsthesia of the face upon the side of the lesion.*

(*b*) *Lesions affecting the descending root of the trigeminus produce anæsthesia of the face upon the side opposite the lesion.*

These conclusions confirm the statement of Meynert, that the descending root of the trigeminus decussates in the pons.

It may be noticed in passing that *a lesion, in order to produce any disturbance of sensibility in the face, must lie in the external lateral part of the formatio reticularis.* In the cases in which the face was not affected, the lesion lay elsewhere than in this portion. In the cases in which it was involved there was anæsthesia of the face.

2. Anæsthesia of the right limbs occurred in cases 3, 4, 6, 7, 14, 17, 20. Anæsthesia of the right arm alone occurred in cases 10, 22. Anæsthesia of the left limbs occurred in cases 5, 11, 12, 13, 16, 18, 19. In all these cases the anæsthesia of the limbs was upon the side opposite to the lesion, although the lesion was situated in all portions of the formatio reticularis from the lower limit of the medulla to the upper border of the pons. *It is therefore evident, first, that in the medulla and pons the sensory tract for each side of the body lies in the opposite half; and, secondly, that there is no decussation of the sensory tracts between the sensory decussation at the lower limit of the medulla and the upper border of the pons.* The facts are therefore opposed to the course of the sensory tracts described by Wernicke and Spitzka, in so far (1) as these tracts are supposed to undergo a second decussation between the sensory decussation and the internal capsule; and (2) as the sensory tracts are supposed to leave the medulla and to pass by way of the cerebellum around the pons. Lesions of the pons in any portion of its vertical extent (*i. e.*, between its cephalad and caudad limits) produce anæsthesia of the op-

posite half of the body. Therefore the sensory tracts must pass through the pons. Therefore they cannot pass through the cerebellar peduncles and the cerebellum unless we suppose that there are two independent sensory tracts, from each side to the cerebrum, a lesion in one of which suspends the function of the other also. It is very possible that some sensory impulses may pass to the cerebellum by the tracts described by Wernicke and Spitzka, and, setting up there a reflex action, be the means of exciting that organ to do its reflex work. But if so these are *not* the sensory impulses which pass to the higher cortical cerebral centres, or which are destined to awake in consciousness a perception of the sensation. The sensations which are perceived consciously are transmitted directly from the surface of the body through the spinal cord, medulla and pons into the internal capsule and thence to the cortical centres, and in their course undergo but one decussation. If that decussation is complete in the cord, the tract remains on the same side from the cord to the capsule. If that decussation does not occur in the cord it takes place in the sensory decussation at the lower part of the medulla.

In all these cases the lesion involved the formatio reticularis of the medulla or pons, and this was the only area of these parts which was affected in *every* case. By the term formatio reticularis I wish to include in the medulla (see fig. 5) the portion lying between the gray matter of the floor of the fourth ventricle and the pyramidal tracts in a dorso-ventrad direction, and between the interolivary tract and the ascending root of the trigeminal nerve in a lateral direction. In as much as there is no case on record in which the olivary body alone was destroyed, it is impossible to state whether it is to be included in the sensory area of the medulla. Its connection with the sensory parts of the cord has led Meynert to consider it a part of the

sensory tract. The question cannot be decided from pathological facts. In the pons the formatio reticularis (see figs. 6, 7) lies between the gray matter of the floor of the fourth ventricle and the lemniscus, in a dorso-ventrad direction, and between the raphé and the external border of the pons, in a lateral direction. There is one case on record (case 26) in which a lesion limited to the inner portion of the formatio reticularis near the raphé in the most cephalad quarter of the pons, and affecting the post. longitudinal bundle chiefly, produced *no* sensory symptoms. It is possible, therefore, that the sensory tracts for tactile sensation no where in medulla or pons approach the raphé, but the number of cases is too few to warrant any general positive statement.

That the sensory tract for the tactile sense lies in the formatio reticularis alone will be more conclusively demonstrated after a review of the other sensory symptoms.

When the cases in which anæsthesia of the face was associated with anæsthesia of the limbs are compared with the cases in which the face alone or the limbs alone were affected, and the situation of the lesion in these three classes of cases are compared, the following diagnostic points can be deduced:

1. *If in any case anæsthesia of one side of the face occurs (not due to neuritis of the trigeminus or to cerebral lesion), the lesion lies in the medulla or pons, in the outer third of the formatio reticularis. Its position in this part is to be determined by the other symptoms present; for, if it is situated high up (cephalad) in the pons, it will be on the side opposite to the anæsthesia, and if it is situated low down (caudad) in the pons or in the medulla, it will be on the same side as the anæsthesia.*

2. *If in any case anæsthesia of the limbs occurs (not due to cerebral lesion), the lesion lies in the medulla or pons, in the inner two thirds of the formatio reticularis, and upon the side opposite to the anæsthesia; or in the spinal cord.*

3. *If one side of the face and the limbs of the opposite side are anæsthetic, the lesion affects the entire lateral extent of the formatio reticularis, and lies in the medulla, or in the pons,* below *the point of union of the ascending and descending roots of the fifth nerve.*

4. *If the face and limbs of the same side are anæsthetic, the lesion lies in the brain at a point* higher than *the junction of the ascending and descending roots of the fifth nerve in the pons. Its position is then to be determined by other symptoms. It may involve the entire formatio reticularis in the upper pons, or crus cerebri; it may be situated in the posterior part of the internal capsule; it may lie in the centrum ovale destroying the radiation of sensory fibres from the internal capsule; it may be in the sensory area of the cortex in which all these traces terminate.*

See Diagram 1.

II. Disturbances of the sensation of pain.

The sense of pain was impaired in cases 4, 5, 6, 9, 10, 11, 19, 20. The subjective sensation of pain was present in cases 19, 21, 24. In a number of the cases no test of this sensation was made. The sensation of pain was found to be normal in cases 1, 2, 7. The distribution of the disturbance of the sensation of pain corresponded in all these cases with the distribution of the anæsthesia, and therefore the conclusions drawn from the disturbances of the tactile sense apply to those of the sense of pain. In the three cases in which the sense of pain was normal the tactile sense was also normal in two, and but slightly affected in the third. In all these three cases the lesion was chiefly in the interolivary tract and lemniscus, and only in the third was the formatio reticularis affected at all. No case of disturbance of the sensation of pain *alone*, accompanied by an autopsy, is on record.

Therefore, until further evidence is brought forward, it

must be accepted that *sensations of pain are transmitted through the formatio reticularis*, and have the same course as tactile sensations.

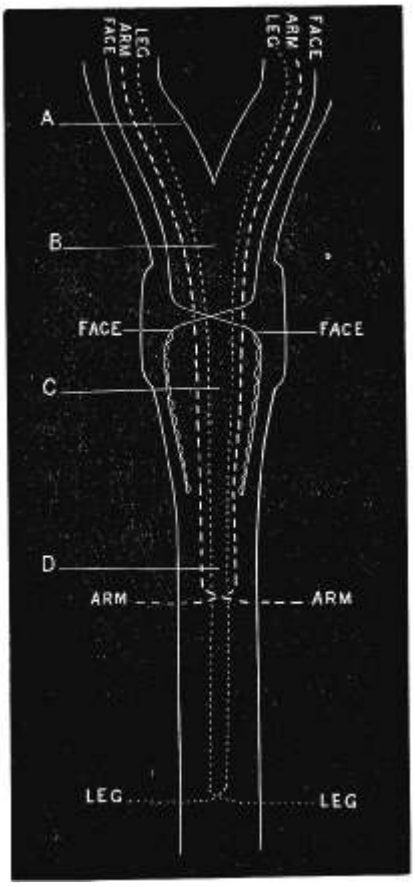

DIAGRAM I.

DIAGRAM OF TRACT CONVEYING TACTILE SENSATION FROM THE SURFACE TO THE INTERNAL CAPSULE.

A.—Lesion in Capsule producing Hemianæsthesia.
B.— " " Crus " "
C.— " " Pons (below upper third) or in Medulla producing Alternating Anæsthesia.
D.—Lesion in Cord producing Spinal Hemianæsthesia.

III. Disturbances of the sensation of temperature.

The sensation of temperature was impaired in cases 4, 5, 6, 7, in all of which cases the disturbance occurred in the

anæsthetic parts. In the remainder of the cases no tests for this sensation were applied. No conclusion can, therefore, be drawn as to the course of the tracts conveying this sensation, although the fact that *disturbances of the sensations of pain and temperature usually occur together* gives a certain probability to the hypothesis that these sensations follow the same tract in both cord, and medulla, and pons.

Subjective sensations of heat and cold were present in a number of cases: of cold, in cases 3, 7, 15, 20; of heat in 6, 10, 24. These subjective sensations had no relation to the anæsthetic parts, but were general, extending to the normal as well as to the affected limbs. In the cases in which the subjective sensation was that of heat, there was also a flushing of the parts in which the heat was felt. It seems probable, therefore, that in these cases the vaso-motor centre in the medulla, the existence of which, in animals, is undoubted, was involved. Foster[1] locates this centre in the medulla in a small area lying just above the calamus scriptorius, and it is a noticeable fact that in these three cases the lesion lay exactly in this region; while in three out of the four cases in which the sensation was one of cold, the same area must have been, to some extent at least, involved, and in two cases (cases 2 and 5) in which there was no subjective sensation, but in which there was other evidence of vaso-motor disturbance, the same area was affected. We have, therefore, eight cases in which *vaso-motor disturbance was associated with a lesion in the upper half of the medulla*, and thirteen cases in which this part was not affected, and in which no vaso-motor disturbance occurred. The *localization of the vaso-motor centre, therefore, which was reached by physiological research is confirmed by pathological observation.*

IV. Disturbances of the muscular sense and consequent ataxia.

[1] "A Text-Book of Physiology." M. Foster, 3d edition, page 218.

Ataxia was present in cases 1, 2, 4, 5, 6, 7, 10, 15, 17, 19, 21, 22, 23, 24.. In cases 1, 2, ataxia was not accompanied by other sensory symptoms. In cases 7, 10, 15, ataxia was marked while the sensory symptoms were slight, in two of these cases the anæsthesia being limited to the face. In the remainder both muscular and tactile senses were affected.

In the cases in which ataxia occurred without affection of tactile sensibility the lesion affected either the interolivary tract in the medulla, or its continuation, the lemniscus, in the pons.

In the cases in which affection of tactile sensibility occurred without ataxia, these parts were not involved in the lesion.

In the cases in which both ataxia and tactile anæsthesia occurred, these parts as well as the formatio reticularis were involved. *The conclusion is warranted that the muscular sense is transmitted along the sensory tracts which lie in the interolivary tract and lemniscus.* In the spinal cord the sensation of muscular sense ascends upon the same side upon which it enters, as we have already seen. But lesions of the interolivary tract and lemniscus produce ataxia in the limbs of the side *opposite to the* lesion. Therefore, *the sensations of muscular sense must decussate in the medulla. They do decussate iu the sensory decussation of the medulla,* as is proven by the facts afforded by cases 4, 5, 7, 8, in which a descending degeneration was traced from a lesion of the lemniscus downward along the interolivary tract to the level of the sensory decussation, and in one case through the sensory decussation to the nuclei gracilis and cuneatus.[1] This course, therefore, corresponds to that of the pyramidal motor tracts.

[1] Kahler (*Praguer med. Woch.*, Jan., 1879) was the first to connect the symptom ataxia with a lesion of the interolivary tract. Meyer (*Arch für Psych.*, Feb., 1882) reported his case without alluding to Kahler. Senator (*Arch. für Psych.*, Oct., 1883) cited both these cases and added one of his own, and ascribed the ataxia to a lesion of the lemniscus as well as of the in-

The sensations of pressure and of the location of a limb are conveyed by the muscular sense and are included under it. In the cases in which they were tested and found wanting, cases 1, 4, 5, 6, 7, the disturbance was limited to the parts which were ataxic.

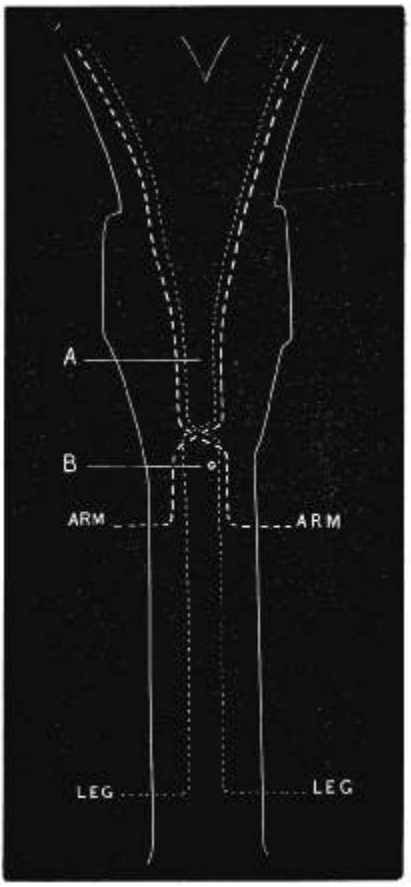

DIAGRAM II.

DIAGRAM OF TRACT CONVEYING MUSCULAR SENSE FROM THE LIMBS.

A.—Lesion above the decussation producing ataxia of opposite side.
B.—Lesion below the decussation producing ataxia of the same side.

terolivary tract. Spitzka. (*Amer Jour. Neurology*, Feb., 1884,) arrived at the same conclusion from his own case and quoted Meyer, but does not allude to Senator. Each of these observers, therefore, may justly claim the independent discovery of the same fact. All the cases hitherto published are here collected for the first time.

The results reached from the analysis of these cases are as follows:

1. The sensory tracts in the medulla are the formatio reticularis and the interolivary tract.

2. The sensory tracts in the pons are the formatio reticularis and the lemniscus.

3. The sensory impulses which have decussated in the spinal cord are conducted along the sensory tract through the medulla pons and crus to the internal capsule without recrossing the median line. These are the sensations of touch, pain, and temperature, and they are transmitted through the formatio reticularis.

4. Those sensory impulses which have not decussated in the spinal cord cross the median line in the sensory decussation of the medulla, and pass upward through the interolivary tract to the lemniscus, in which they ascend to the internal capsule. These are the sensations included under the term muscular sense.

5. If any sensory impulses pass to the cerebellum, they are only those whose result is to awaken reflex action, and they are not the sensory impulses whose reception in the cortical cells is capable of awakening a conscious perception of the sensation. The sensations consciously perceived do not pass through the cerebellum on their way to the cerebrum.

The course of the sensory tracts thus established by pathological cases is the same as that which has been determined by the anatomical and embryological researches of Flechsig, by the one experiment made in accordance with the method of Gudden, and by the investigation of the microcephalic brains in which the motor tracts were absent. It may therefore be accepted as the only one possible in the medulla and pons.

But if it be accepted that the muscular sense passes

along the interolivary tract and decussates in the medulla, while no other sensations so decussate, we may trace this tract downward into the cord, and thus determine the function of a portion of the cord. The interolivary tract is made up partly of fibres from the nucleus cuneatus and partly from fibres from the nucleus gracilis, and these are respectively the terminal stations of the columns of Burdach and of Goll. It is therefore probable that in these columns the muscular sense is transmitted, the column of Goll transmitting this sense from the legs, and the column of Burdach transmitting it from the arms. This hypothesis is substantiated by the fact that these are the columns which are diseased in locomotor ataxia, in which the most marked and constant symptom is a loss of the muscular sense. This does not exclude a transmission of touch also in these columns, since both columns are connected with the formatio reticularis, as well as with the interolivary tract. It makes it probable that Schiff's conclusions regarding animals are true as regards man, and that *in the spinal cord sensations of touch and of the muscular sense pass up in the posterior columns*, while sensations of temperature and pain pass up in other sensory tracts—viz., in the gray matter or in the direct cerebellar columns.

But it is improbable that sensations of pain and temperature pass in the direct cerebellar columns: first, because these columns only commence in the cord above the level of the first lumbar nerve at the level of the Clarke column of gray cells in the posterior median gray matter; and secondly, because these columns go to the cerebellum, while lesions of the cerebellum do not interfere with sensations of pain and heat or cold, while, on the other hand, such sensations are affected in diseases of the pons, through which the direct cerebellar columns do not pass. For these reasons it is probable that *sensations of temperature and pain pass up in the gray matter of the spinal cord.*

What, then, remains as a function for the direct cerebellar columns? I am not aware that any hypothesis has been offered, and where proofs are wanting, hypotheses are worth little. But anatomy and pathology combined seem to indicate a possible function for these columns, as follows:

1. The columns arise (Flechsig) from a set of cells (the Clarke column) whose extent is coextensive with the entrance into the cord of nerves which come from the thoracic and abdominal viscera, and which form a centripetal path for impulses originating within the great cavities of the body. 2. Diseases in the cord which interfere with the function of the direct cerebellar columns (viz.: transverse myelitis—myelitis of any kind) are attended by an irregular action of the organs within the great cavities, especially within the abdomen. The gastric crisis of locomotor ataxia and the habitual constipation of myelitis are examples in point. 3. Diseases of the cerebellum are known to give rise to disturbances of function of the viscera of the great cavities—especially of the abdominal viscera; indigestion, vomiting of a peculiar kind, obstinate constipation, polyurea, albuminuria, etc., being well-known symptoms of cerebellar disease.[1]

For these reasons I would advance the hypothesis that among other functions of the cerebellum that of a reflex centre for the proper regulation of the functions of vegetative life is one, and further that the *path of impulses between the vegetative organs and this reflex centre lies in the direct cerebellar columns of the spinal cord* as far as centripetal impulses are concerned.[2]

[1] It is a noticeable fact that these symptoms of cerebellar disease, *unlike the ataxia*, are not produced by lesions of the vermiform lobe *alone*, but occur when the cerebellar hemispheres are the seat of lesions, such as abscess and softening from embolism and thrombosis, whieh neither increase the intra-cranial pressure nor affect in any way directly the floor of the fourth ventricle and the pneumogastric centres there or in the flocculas.

[2] The cases here collected present many interesting features aside from those

SENSORY TRACTS IN THE CRURA CEREBRI.

The formatio reticularis and lemniscus continue upward through the tegmentum of the crus lying external to the red nucleus, and pass into the posterior part of the internal capsule. It is the opinion of Flechsig that the lateral lemniscus passes through the internal capsule into the corona radiata of the tegmentum and thus directly to the cortex. Roller, Forel, and Wernicke trace it in part at least to the laminæ medullares of the optic thalamus and make it end in this ganglion. Fibres from the formatio reticularis pass

concerning the sensory tract. The discussion of these symptoms, and of the light which they throw upon the normal functions of the medulla and pons, cannot be entered upon here, as these are subjects aside from the present purpose. The symptoms are, however, analyzed, and are reserved for consideration at another time.

1. Paralysis of the iii. nerve, right. Case 5.
" " " left. Cases 9, 14, 15.
2. Paralysis of the vi. nerve, right. Cases 5, 11, 19, 26.
" " " left. Cases 1, 4, 14, 15, 21.
3. Conjugate deviation of the eyes to the right. Cases 6, 9, 26.
" " " " " left. Cases 5, 17.
4. Ptosis. Cases 7, 9, 15.
5. Nystagmus. Cases 1, 4.
6. Diplopia. Cases 4, 6, 9, 19, 21.
7. Myosis. Case 15.
8. Mydriasis. Cases 7, 9.
9. Dimness of vision. Cases 7, 11, 17, 21, 23, 24.
10. Inflamation of the cornea. Cases 5, 10, 14.
11. Paralysis of vii. nerve, right. Cases 4, 5, 11, 12, 15, 17, 18, 20, 23.
" " " " left. Cases 6, 9, 10, 14, 17, 19, 22,23, 24, 25.
12. Trismus. Cases 9, 10, 17.
13. Deafness in the right ear in cases 10, 12 ; in the left ear in cases 9, 14, 24.
14. Tinnitus aurium in case 5.
15. Difficulty in swallowing in cases 2, 3, 5, 6, 7, 11, 12, 14, 16, 22, 24.
16. Difficulty in speaking, not aphasia, in cases 2, 3, 4, 7, 10, 11, 12, 15, 16, 18, 22, 23.
17. Paralysis of the XII. nerve, right, in cases 2, 4, 6, 7, 10, 12, 20.
" " " " left in cases 2, 5, 6, 11, 16, 18, 24, 25.
18. Paralysis of arm and leg, right, in cases 6, 10, 14, 15, 17, 20, 21, 22, 23.
" " " left, in cases 5, 11, 14, 16, 18, 19, 24.
19. Increased knee-tendon reflex, right, in cases 6, 7.
" " " left, in case 5.
20. Absence of the skin reflexes, right, in cases 6, 7.
" " " left, in case 5.

Situation of the Lesion.

On the right side in cases 2, 5, 11, 12, 13, 15, 16, 18, 19, 23.
" left " " 1, 2, 3, 4, 6, 7, 8, 9, 10, 14, 17, 20, 21, 22, 24, 25.
On both sides in case 2.
In the medulla alone in cases 1, 2, 3, 6, 9, 24.
In the pons alone in cases 10, 11, 13, 14, 15, 16, 17, 19, 20, 21, 22, 25, 26.
In both medulla and pons in cases 4, 5, 7, 8, 12.

to the substantia nigra of the crus and end in it. Others pass to the laminæ medullares of the optic thalamus and end in it. Others still unite with fibres from the red nucleus, and leave the tegmentum in a large bundle which passes through the internal capsule in its posterior part and radiates toward the cortex (the Haubenstralung of Flechsig). No attempt has been made to assign functions to the various bundles of fibres issuing from the tegmentum.

Cases of lesion of the crus are rare, and those on record are chiefly lesions of the pes and not of the tegmentum. Nothnagel was able to cite but nine cases, and his conclusion as to the sensory tracts is merely negative. They do not lie in the inner two thirds of the pes, as lesions there give rise to motor symptoms only.

Two cases are on record in which the red nucleus has been diseased, and in neither of these was sensation affected.[1] The red nucleus is in anatomical connection with the opposite superior cerebellar peduncle, as is proven by the fact that atrophy of one cerebellar hemisphere is accompanied by atrophy of the opposite red nucleus. Lesions of the corpora quadrigemina usually involve the red nucleus, and in these there is no disturbance of general sensation.[2] Such lesions may produce incoördination which indicates a functional relation between the red nucleus and the cerebellum. There is, therefore, no reason to assign sensory functions to the red nucleus, or to suppose that the sensory tracts pass in the inner half of the tegmentum.

It is not probable that the sensory tracts pass in the outer third of the pes, for this part consists of fibres which connect the cerebrum and cerebellum, and is absent when either the cerebrum or cerebellum is absent.[3]

[1] Kahler: *Arch. f. Psych.*, x. Kahler and Pick: *Zeitsch. f. Heilk.*, ii., p. 305.

[2] See Wernicke: "Lehrbuch d. Gehirn.," vols. ii. and iii., sections on Corp. Quad.

[3] Compare Flechsig's case of deficient cerebellum with my case of deficient cerebrum, in both of which this part was absent.

It is therefore probable that in the crura the sensory tracts lie in the outer half of the tegmentum, in which part lie the formatio reticularis and lemniscus, which have been shown to be the sensory tracts in the pons. There are no pathological facts to prove or to oppose this hypothesis. It is reached by a method of exclusion, but until further facts are offered may be accepted.

SENSORY TRACTS IN THE BRAIN.

I.—*Anatomical.*

The formatio reticularis terminates at the upper level of the crura, and from this point fibres pass into the posterior third of the internal capsule. Into this part of the capsule the lemniscus also passes. These tracts here become indistinguishable, but are known to lie in the internal capsule, as they did in the pons, posterior to the motor tract. It is well-known that lesions of the posterior part of the internal capsule produce anæsthesia of the opposite side of the body; a pathological fact which confirms the course of the sensory tract through this part.

At the upper level of the internal capsule its fibres radiate toward the cortex. The point of radiation of the fibres which form the sensory tract is known as the "carrefour sensitif." This is just opposite the middle of the optic thalamus and at its upper level. Here, too, lesions produce hemianæsthesia.

Above the "carrefour sensitif" the nerve fibres from the capsule become so mingled with radiating fibres from the optic thalamus that in an adult brain they cannot be distinguished. Flechsig has found, however, that the former obtain their medullary sheaths at an earlier date than the latter in fœtal life. He was thus able to trace the sensory tract. He affirms that it approaches the posterior and inner border of the motor tract, and in the corona radiata passes to the cortex of the parietal lobe where it ends in

the convolutions lying posterior to the fissure of Rolando, and anterior to the occipital lobe of the brain.

From a review of the facts just stated it is evident that a direct anatomical connection can be traced from the surface of the body to the parietal cortex of the brain, by means of the sensory nerves, the sensory nerve-tracts in the cord, medulla, and pons, and in the interior of the brain itself.

It is admitted that no one nerve fibre can be followed from a point on the surface to a point in the brain. In fact it seems to be a law of nervous transmission that impulses are always modified in their course by passing into and then issuing from nerve cells. In the course of sensory impulses the cells in the gray matter of the cord, and probably those in the gray matter of the medulla, crus, and optic thalamus are traversed by some, if not by all of them. But however such cells may modify the impulse they do not interrupt it, and their presence does not affect the fact of a connection between the surface of the body and the cortex of the brain. The most considerable collection of cells in this tract is the optic thalamus, and for many years it has been thought to take a large part in the reception and transmission of sensory impressions. Recent investigation of cases of thalamus disease by Nothnagel[1] and Wernicke[2] has thrown some doubt upon its sensory function, as far as the sensations of touch, temperature, and pain are concerned. And it is still an open question whether the sensory symptoms occurring in its diseases are due to an implication of sensory centres, or to an affection of the sensory tracts passing along its periphery in the internal capsule. Until further facts are offered, however, the conclusions of Flechsig must be accepted, since these are the only ones well established and of value.

Anatomical study, pursued by the various methods of in-

[1] Nothnagel: "Topische diagnostic d. Gehirnk."

[2] Wernicke: *l. c.*, vol. ii. and iii.

vestigation at present known, leads, therefore, to the conclusion that the tracts conveying sensations of touch, pain, temperature, and the muscular sense, pass to the parietal region of the cortex of the hemispheres.

It is not known whether *all* the sensations from one side of the body pass to the opposite hemisphere of the brain. The pathological facts here cited would indicate that they do. It must be remembered, however, that for many years, and until a very recent date, it was supposed that a lesion of one hemisphere produced a paralysis upon the opposite side of the body *alone*. It is now admitted that, in as much as each hemisphere is connected with both sides of the body by motor tracts—the larger of the motor tracts decussating, and the smaller not decussating in the medulla,—a lesion of one hemisphere produces paralysis upon the opposite side of the body, and a certain amount of weakness upon the same side of the body. It has recently been established that a lesion of one hemisphere in the visual area produces, not blindness in the opposite eye as was formerly supposed, but a certain degree of blindness in both eyes, that in the opposite eye being greater in extent than that in the eye of the same side.[1] The olfactory fibres pass from each bulb to both hemispheres. The argument from analogy would therefore indicate that, as regards other sensations, each half of the brain is in relation with both sides of the body, its connection with the opposite side being more extensive than that with the same side. The probability therefore is, that *all* the sensations from one side of the body do *not* pass to the parietal cortex of the opposite side; but that while the majority so pass, a portion go up to the cortex of the same side from which they come. This probability is somewhat strengthend by the fact that

[1] See Mauthner: "Vorträge aus des Gesammtgebiet der Augenheilkunde." "Gehirn und Auge," Wien, 1883. Also Wernicke: *l. c.*, vol. i., p. 69. Also Starr: *Amer. Jour. Med. Science*, Jan., 1884.

complete hemianæsthesia from cortical disease is far less common than complete hemiplegia, a fact which would find an easy explanation if it could be shown that both sides of the body were connected by sensory tracts with each half of the brain. A more careful and accurate examination of future cases may furnish facts to decide this question. At present it can only be stated that a connection has been established between the sensory tracts and the parietal regions of the brain.

II.—*Physiological.*

The researches of physiologists in determining the functions of the brain have been productive of the most successful and brilliant results ever achieved in that department of science. From the beginning of investigation of this subject by Fritsch and Hitzig in 1870 to the latest experiments of Munk in the present year, a continuous succession of interesting and valuable facts have been discovered. This is not the place to trace the history of these discoveries, or to give in detail the methods pursued. The results which bear upon the subject in hand—the location of the sensory areas in the cortex—must be considered briefly.

Ferrier was the first to attempt to determine the sensory area of the cortex. Recognizing the fact that in animals no reliable information of sensory disturbance due to *irritation* of the cortex could be obtained, he resorted to the method of extirpating various areas of the cortex with a view of producing anæsthesia. As the result of several experiments, one of which is given in detail,[1] he concluded that the tactile centre was to be found in the hippocampus major and the uncinate convolution, in the monkey. As he admits that other regions, especially the occipital cortex and part of the corona radiata, were injured in the course of the experiment, the result cannot be accepted as con-

[1] Ferrier: "Functions of the Brain," p. 179.

clusive. That, in the monkeys operated on, tactile sense was destroyed, may be admitted, though it is not stated whether the loss of tactile sense was permanent or only temporary. But since, in destroying this sense, several regions of the brain were destroyed and the internal capsule was injured, it seems somewhat arbitrary to limit the function to one of the regions destroyed. That the tactile centre in man lies in the hippocampal region cannot be admitted; for, first, we have already seen that the sensory fibres do not pass to this region; and second, we shall see, in the pathological section, that lesions of this region do not produce anæsthesia.

The conclusion of Ferrier therefore stands without anatomical or pathological support.

It is probably from a repetition of his earlier experiments that Ferrier has been recently led to change his opinion. In his last announcement (Nov., 1883, Oration before the Med. and Chirurg. Society, London, rep. in *Brit. Med. Jour.*, Nov. 30, 1883) he says: "I have all along held, and hold, both on experimental and on clinical grounds, that the centres of common sensation, including muscular sensibility, are anatomically distinct from those of motion, and are situated in the subcortical region." He thus retracts the statement that sensory centres are situated in the hippocampal region and refuses to locate them on the cortex at all.

The experiments of Munk are more numerous, more carefully conducted, and more complete in observation and description, and his conclusions conform to both anatomical and pathological facts. His method is to extirpate limited areas of the cortex in various regions, and after the animal has entirely recovered from the temporary effects of the operation, to determine what functions are impaired. The animals used were dogs and monkeys. Munk has found

that the area of common sensation (Fühlsphäre)—including sensation of pressure, location of a limb, muscular sense, and touch, lies in the central region, including the anterior and posterior central convolutions, and in the adjacent portions of the cortex. He divides this area into special regions, for the hind leg, fore leg, head, eye, and ear muscles, neck and body. These regions coincide with, but are somewhat more extensive than, the corresponding motor centres for these parts. Destruction of any one of these regions, therefore, produces paralysis and anæsthesia in the parts with which it is connected. The paralysis Munk ascribes to the loss of the memory of the combination of impulses necessary to produce the desired motion (innervationsgefuhl and bewegungsvorstellungen). The anæsthesia persists for ten weeks after the extirpation of a single centre, after which time the animal recovers, the adjacent region taking up the function of the one destroyed. If the entire area, however, is extirpated, the anæsthesia is permanent and complete. To produce complete anæsthesia the entire parietal cortex and a portion of the frontal cortex, must be destroyed. The sensory portion of the cortex is therefore assigned by Munk to the parietal area and central region of the brain.[1] This conclusion is in harmony with the anatomical fact already stated, that this portion receives the sensory tracts.

The experiments of Goltz,[2] though interpreted by him as contradictory to the theory of localization, really substantiate indirectly the conclusion of Munk as opposed to that of Ferrier.

Goltz extirpated the greater part of the external convex surface of the hemispheres, leaving the portions at the base of the brain intact. He thus destroyed Munk's Fühlsphäre

[1] Munk's conclusions are to be gathered from his reports to the Physiol. Soc, of Berlin, to be found in *Pfluger's Arch. f. Physiologie.*

[2] Goltz: "Verrichtungen des Gehirns."

but did not injure the hippocampal region—Ferrier's area of touch. According to his description of the animals thus experimented upon, there was at first a decided loss of sensation to touch, pain, and the muscular sense, though he claims that this was not complete, and that after a time the animal recovered. While his experimental results are interpreted by him as contradicting the results of other experiments in favor of localization, it is evident that, as regards this special subject of the localization of the sensation of touch, his results are in accord with those of Munk and are opposed to those of Ferrier.[1]

The most recent experiments to determine the sensory centres of the cortex are those of Tripier, of Montpellier, France, reported in the *Revue Mensuelle de Médecine*, 1880–1881. Like other experimenters, Tripier has extirpated various areas of the cortex, and observed the results. He reaches the same conclusion as Munk, and affirms the existence of sensory centres on the so-called motor area. He finds that the sensory area for a definite limb coincides with, but is more extensive than, the motor area for the same limb. He thus arrives at a similar arrangement of sensory areas to that already described by Munk.

At the same time that Tripier was working in France, Moeli in Berlin was investigating the same subject. His results are published in *Virchow's Arch.*, Bd. 76, and coincide with those of Munk and Tripier.

Thus three experimenters, working independently, arrived at the same time at a similar conclusion, viz.: that the motor and sensory areas of the brain for any one limb coincide.

But physiological experiment, however precise, affords information concerning the functions of the brain in animals only. It is solely by means of the study of clinical cases

[1] This is not the place to discuss the merits of Goltz' objections to localization. The reader is referred to the *Journal of Physiology*, Dec., 1883, for a complete review and careful criticism of the respective results of Goltz and Ferrier.

that results can be reached in the case of man. The final appeal must, therefore, be made to pathology.

III.—*Pathological.*

Are there any cases on record in which the occurrence of sensory symptoms during life must be ascribed to a limited cortical lesion discovered at an autopsy and accurately localized? When Nothnagel wrote his great work "Topische diagnostic der Gehirnkrankheiten," in 1879, he was obliged to say: "Disturbances of sensation have as yet no bearing upon the diagnosis of cortical lesions." The stimulus given to the observation of brain lesions by Nothnagel's work was productive of many careful records of cases, and in 1880 Exner was able to find twenty two cases in the journals, in which sensory disturbance had been associated with cortical disease. In all of these cases he found the lesion to be within or very near to the central or motor region.[1] No general collection of cases of cortical lesion has been made since that of Exner; the cases collected by Charcot (*Rev. de Méd.*, 1883) being studied solely from the point of view of motor symptoms, and no mention being made of the condition of sensation in the majority of the cases. Exner did not have access to American journals when making his collection. I have, therefore, examined the American journals for the past twenty years, and the chief European journals published between Jan., 1880, and Jan., 1884, and have found a large number of cases in which sensory symptoms have been noticed, and in which a *post-mortem* examination has shown a lesion in the cortex of the brain. It is to the study of these cases that we at once proceed, the cases being first cited and then analyzed.

In the cases chosen there was but *one lesion of limited extent*, situated *in the cortex* of the brain, and *not affecting the*

[1] In sixteen of Exner's cases the central convolutions were actually involved. In the others the parietal convolutions near the post-central conv. were affected.

basal gauglia. These are the only cases from which conclusions can be legitimately drawn, and therefore all others are ruled out from consideration, although a large number were found in which sensory symptoms were associated with extensive or multiple lesions of the cortex.[1]

ANALYSIS OF THE CASES.

In all the cases here collected sensory symptoms were present. In all these cases an autopsy showed a diseased condition, limited to the cortex of the hemisphere of the opposite side, in the anterior or posterior central convolutions, or in the convolutions of the parietal lobules,[2] all other parts of the brain being normal.

The sensory symptoms were of different kinds, which may be classified into (1) symptoms of irritation of the cortex, including hyperæsthesia, hyperalgesia, paræsthesiæ, and subjective pain, and (2) symptoms of destruction of the cortex, including anæsthesia, analgesia, and ataxia. In a number of the cases, the first class of symptoms were succeeded by the second class in the course of the disease.

1. Sensory symptoms indicating irritation of the cortex.

These occurred in fourteen cases. In some the irritation was temporary, occurring just before or just after an epileptic convulsion. In these cases the condition was one of cortical epilepsy, and the diagnosis was confirmed by the autopsy, the local lesion being found in that part of the motor area corresponding to the muscles which first became convulsed. It is therefore evident that the sensory aura of cortical epilepsy may be brought into connection with cortical disease in the sensory-motor area of the brain.

In some of the cases the irritation was permanent, being due to disturbances of circulation in the cortex set up by a

[1] The American cases in this collection are cited by me in full in the *Amer. Jour. of the Med. Sci.* for July, 1884.

[2] For convenience and to avoid repetition, these convolutions are included in the term "*sensory-motor area.*"

Case.	Sex.	Age.	Symptoms.	Duration.	Lesion.	Situation.	Reported by.
1	M.	67	Anæsthesia l. face and arm.	4 mos.	Softening from thrombosis.	A. C., P. C.; m. 1-3.	Petrina: *Zeit. f. Heil.*, ii., 388.
2	M.	30	" r. " "	3 mos.	Hemorrhage.	Sylvian Reg. A. C. l. 1-3.	" " ii., 388.
3	M.	33	Analgesia r. " "	3 wks.	Tubercle.	3 F. A. C. l. 1-3.	" " ii., 388.
4	F.	39	Paræsthesia r. " "	4 mos.	Gumma.	P. C.; l. m. 1-3.	Sands: *Med. News*, April, 1883.
5	F.	20	Analgesia r. " and body.	3 wks.	Softening.	A. C.; l. 1-3 3 F.	Petrina, *l. c.*
6	M.	28	Paræsthesia of arm.	2 yrs.	Softening.	P. C. m. 1-3; Ang. Gy.	Dreshfeld: *Practitioner*, May,'75.
7	M.	28	" "	2 yrs.	Tubercle.	A. C., P. C.; m. 1-3.	Edinger: *Arch. Psych.*, x., 93.
8	F.	18	Hyperæsthesia of r. arm and leg.	5 mos.	Sarcoma.	Sup. Par. Lob.	Monakow: " " xi., 626.
9	M.	28	Anæsthesia of r. arm.	10 days.	Abscess.	A. C., P. C.; m. 1-3.	Bender: *Deut. med. Woch.*, No. 50, 1882.
10	M.	27	" l. "	1 mo.	Tubercle.	A. C., P. C.	Bramwell: *Edin. Jour.*, xxiv.,145.
11	F.	56	" l. "	4 yrs.	Cyst softening.	Inf. Par. Lob. P. C.; m. 1-3.	Carter: *Med. Times and Gaz.*, ii., 399, 1880.
12	F.	60	" r. "	1½ yrs.	Old clot.	3 F.; A.C., l. 1-3, Is. Reil.	Wood: *Phil. Times*, v., 470.
13	M.	34	" r. "	3 mos.	Abscess.	Inf. Par. Lob. P. C.; m. 1-3.	Cock: *Amer. Jour.*, Oct., 1852.
14	M.	19	" l. "	1½ mos.	Softening.	A. C.; m. 1-3.	Noyes: " July, 1882.
15	M.	50	Paræsthesia of l. arm and leg.	4 mos.	Sarcoma.	A. C., P. C.; m. u. 1-3.	Peabody: *Arch. Med.*, April, '82.
16	F.	27	" l. " "	2 days.	Softening from thrombosis.	A. C., P. C.; m. u. 1-3	Bumstead: " Oct., '79.
17	M.	53	" and ataxia of l. arm and leg.	7 mos.	Tubercle.	Syl. Reg. Isl. Reil.	Seaman: *Phil. News*, Jan., 1883.
18	M.	54	Anæsthesia of r. arm and leg.	6 mos.	Abscess.	A. C., P. C.; m. u. 1-3.	Page: *Med. & Surg. Rep.*, xxi., 29.
19	M.	35	Paræsthesia of l. arm and leg.	2 yrs.	Gumma.	A. C., P. C.; m. u. 1-3.	Morton: *Chic. Jour. & Exam.*, xlvi., 21.
20	F.	38	Hyperæsthesia of l. " "	Not stated.	Gumma.	A. C., P. C.; u. 1-3.	Mills: *Arch. Med.*, Aug. 1882.
21	M.	57	Hyperæsthesia and anæsthesia of l. arm and leg.	"Some months."	Tubercle.	Sup. Par. Lob.	Petrina, *l. c.*
22	F.	2¾	Anæsthesia r. arm and leg.	2 days.	Tub. meningitis.	A. C., P. C.; Par. Lob.	Cerf: *Arch. klin. Med.*, xxxi.,431.
23	F.	2½	" r. " "	23 days.	Abscess.	Sup. Par. Lob.	Smith: Jour. Ment. and Nerv. Dis., July, 1880.
24	M.	66	" r. " "	18 mos.	Softening.	A. C., P. C.; m.l. 1-3 Inf. Par. Lob.	Mills: *Phil. Times*, ix., 246.
25	M.	—	" r. " "	28 days.	Abscess.	P. C. mid. 1-3 Inf. Par. Lob.	Liddell: *Amer. Jour. Med. Sc.*, July, 1883.
26	M.	36	" and ataxia l. arm and leg.	2 mos.	Softening.	P. C. and Inf. Par. Lob.	Carson: *Practitioner*, xv., 217.
27	F.	30	" l. arm and leg.	10 mos.	Carcinoma.	P. C. m. 1-3; Inf. Par. Lob.	Mills: *Arch. Med.*, Aug., 1881.
28	F.	53	" l. " "	7 mos.	Glioma.	A. C., P. C.; mid. 1-3.	Wood: *Amer. Jour. Med. Sc.*, April, 1864.
29	M.	—	L. hemianæsthesia.	1 yr.	Sarcoma.	A. C., P. C.; u. 1-3 Sup. Par. Lob.	Seguin: *Trans. Neuro. Soc.*, 1877.
30	F.	27	L. "	2 mos.	Sarcoma.	A. C., P. C.; m. u. 1-3.	Bell: *Amer. Jour.*, July, 1870.
31	M.	30	L. "	6 mos.	Tumor.	A. C., P. C.; m. u. 1-3.	Janeway: *Med. Record*, ix., 651.
32	M.	33	R. "	1¼ mos.	Abscess.	P. C., Inf. Par. Lob.	*Hospital Gazette*, vi., 552.
33	M.	50	R. "	3 mos.	Softening from thrombosis.	A. C. l. 1-3; Is. Reil.	Petrina, *l. c.*
34	M.	67	R. "	22 days.	Softening.	A. C., P. C. l. 1-3; Is. Reil.	Tripier: *Rev. Mens.*, 1880, p. 138.
35	F.	57	R. "	20 days.	Softening.	A. C., mid. 1-3.	" " 1880, p. 135.
36	F.	23	R. "	1 mo.	Softening from emb.	A. C., P.C. m.l. 1-3; Inf. Par. Lob.	Dejerine: *Prog. Méd.*, viii., 781.
37	F.	42	R. hyperalgesia.	3 days.	Glioma.	A. C., P. C., l. 1-3.	Cheesman: *Arch. Med.*, Aug., '81.
38	M.	66	R. hemianæsthesia.	4 mos.	Softening.	A. C., P. C. l. 1-3; Par. Lob.	Mills: *Med. Bulletin*, i., 13.
39	F.	51	R. "	2 yrs.	Clot.	Is. Reil.; Inf. Par. Lob.	Richardson: *Richmond Med. Jour.*, iii., 426.
40	M.	72	R. "	20 yrs.	Hemorrhagic cyst.	A. C., P. C.; Is. Reil.	Starr: *Amer. Jour.*, July, 1884.
41	M.	42	R. "	Not stated.	Clot.	Inf. Par. Lob.	Tripier, *l. c.*

[1] These cases were cited in full in the original essay. They are here tabulated to save space.

Abbreviations.—A. C., P. C.—Ant. and Post-Central Convolutions; l., m., u.—lower, middle, upper thirds of these convolutions; Sup. Par. Lob., Inf. Par. Lob.—Superior and Inferior Parietal Lobules; Is. R.—Island of Reil. 3 F.—third Frontal Convolution.

lesion (embolism, thrombosis, hemorrhage), or to pressure upon the cortex by a tumor or clot. It was in these cases that the lesion, after for a time irritating the cortex, produced its disintegration, and the symptoms of irritation were followed by those of destruction.

2. Symptoms of destruction of the cortex.

These occurred in thirty-two cases. In all of these cases the loss of sensation was permanent, but in none of them was it complete. This fact adds probability to the hypothesis already advanced, that *each* side of the body is connected by sensory tracts with *both* sides of the brain, destruction of the sensory area of one half of the brain producing great impairment, but no absolute loss of sensation, in the opposite side of the body. This hypothesis also offers an explanation for some of the numerous cases which are on record, in which a careless examination of the condition of sensation failed to detect any anæsthesia, but in which a disease of the sensory-motor area of the brain was discovered after death. There are many such cases to be found in medical literature, and Ferrier has cited them in opposition to the views of Exner, who, as already stated, was the first to connect sensory symptoms with lesions of the motor area. Such cases are, however, of little value, since in many of them no mention is made of the condition of sensation, and in many the tests applied to detect the various kinds of disturbance of sensation were not accurately applied. Like the cases cited by Brown-Séquard in opposition to all the facts of localization, these cases do not bear a strict examination.

The forty-one cases here collected, together with the twenty-two cases collected by Exner, afford sufficient ground for the conclusion that lesions affecting the cortex of the brain in the central and parietal convolutions, may give rise to sensory symptoms.

It is well known that definite regions of the central convolutions of each hemisphere govern definite portions of the muscular system. The motor area for the opposite half of the face and tongue lies in the lower third of the central convolutions. The motor area for the opposite arm lies in the middle third of the central convolutions. The motor area for the opposite leg lies in the upper third of the central convolutions, including the paracentral lobule.[1]

It is interesting to find that a similar distribution of the sensory areas may be affirmed. In the cases here collected, the lesion lay in the lower third of the sensory motor area when the face was affected by sensory disturbances; the lesion lay in the middle third of this area when the arm was affected; the lesion lay in the upper third of this area when the leg was affected. When sensory symptoms occurred in both face and arm, the lower and middle areas, or their junction, were found diseased. When sensory symptoms occurred in both arm and leg, the middle and upper areas, or their junction, were found diseased. And it is a noticeable fact that in no case were face and leg affected together without implication of the arm, a fact which affords a clear indication that their areas are separated by that ofthe arm.

It is therefore justifiable to conclude that:

1. In the cortex of the brain sensations of touch, pain, temperature, and the muscular sense are perceived.

2. These perceptions occur in the gray matter of the anterior and posterior central convolutions and of the parietal convolutions; sensations from one side of the body being perceived in the opposite half of the brain in a more intense degree than in the same half of the brain.

[1] See Charcot: "Localization des Maladies Cérébrales," Paris, 1876; and collections of cases in the *Rev. de Méd*, 1877, 1879, and 1883.
Nothnagel, "Topische diagnostic der Gehirnkrankeiten," 1879.
Exner, "Untersuchungen ub. d. Local. d. Functionen in d. Grosshirnrinde," 1880.
Wernicke, *l. c.* Ferrier, "Localization of Functions in the Brain," 1878.
Starr, *Amer. Jour. Med. Sci.*, April and July, 1884.

3. The various sensory areas for various parts of the body lie about and coincide to some extent with the various motor areas for similar parts—the area for the face, arm, and leg, lying in the lower, middle, and upper thirds of the sensory-motor region respectively.

4. While the motor area is confined to the central convolutions, the sensory area includes to some extent the convolutions of the parietal lobe which lie adjacent and posterior to them. It is therefore more extensive than the motor area.

These conclusions are further strengthened by a review of the cases of lesion of other areas of the cortex. Such cases cannot be cited here.[1] It may, however, be affirmed that in cases of disease limited to the frontal, temporal, sphenoidal, or occipital regions of the brain disturbance of sensation is not observed.[2]

GENERAL CONCLUSIONS.

A study of the anatomy of the central nervous system by the various methods at present used, an examination of embryological facts both in normal infants and in cases of abnormal development, a review of the results of physiological experiment and a collection of pathological cases of small lesion limited to definite tracts and to definite areas of the cortex of the brain, have led to the following conclusions:

A.—The surface of the body is connected with a definite region of the surface of the brain by distinct tracts which convey sensory impressions. These impressions enter the spinal cord by the posterior nerve-roots, and then ascend in different tracts.

(*a*) The impressions destined to awaken the sensation of

[1] See authorities cited in last note.

[2] This is also the result reached from a study of the American cases which I have collected and published in the *Amer. Jour. of Med. Sci.*, April and July, 1884.

touch pass at once to the opposite half of the spinal cord, to a great extent if not entirely, and ascend in the posterior white columns, the impressions from the legs passing in the posterior median, and those from the arms in the posterior lateral columns. On reaching the medulla these impressions pass to the formatio reticularis of the same side on which they were in the cord, and ascending in this tract through the pons and crus, reach the internal capsule, where they are conducted by a tract lying in the inner half of its posterior third to the corona radiata, whence they diverge to the cortex of the central and parietal regions, the impressions from the leg being perceived in the upper third of these regions, and those from the arm being perceived in the middle third of these regions.

(*b*) The impressions destined to awaken the sensations of pain and temperature also cross the median line immediately after entering the spinal cord, and pass up in the gray matter of the cord, probably in its posterior inner part. On reaching the medulla they enter the formatio reticularis, and from this point their course is identical with that of the impressions of touch already traced.

(*c*) The impressions destined to awaken the sensation of the location and of the motion of a limb ascend in the spinal cord, in the posterior white columns of the same side upon which they enter; the muscular sense from the legs passing up in the posterior median, and that from the arms in the posterior lateral columns. On reaching the medulla, these impressions pass to the opposite side through the sensory decussation, then ascend in the interolivary tract, to the pons, where they enter the lemniscus, and gradually turn outward from the median line as they pass up through the pons. In the crus, these impressions are conducted by the lateral lemniscus, which lies in the outer third of the tegmentum, and which passes directly into the internal capsule,

there being situated in the middle part of the posterior third, external and in close approximation to the sensory tract of touch already described. From this point, the course of these kinds of impressions cannot be distinguished from one another, and their termination is in the cortex of the central and parietal regions, the muscular sense of the leg being perceived in the upper third, and that of the arm in the middle third, of these regions.

B.—Sensory impressions from the face enter the pons by the sensory root of the trigeminus, and pass downward to sensory cells which lie in the lateral portion of the formatio reticularis, and which are arranged in a column extending from the junction of the upper and middle third of the pons, to the lower limit of the medulla. The upper portion of this column receives the fibres from the upper branch of the nerve; the middle portion from the middle branch, and the lower portion from the lower branch. The course of sensory impulses, from these sensory cells to the brain, is in the lateral part of the formatio reticularis of the same side upon which they enter, up to the junction of the upper and middle thirds of the pons, where they cross the median line and join the sensory impressions from the body in the formatio reticularis. In the crus cerebri, these impressions pass in the outer half of the tegmentum, and, thence entering the posterior third of the internal capsule, pass on to the corona radiata. In the divergence of fibres in the corona, these impressions pass along the lowest radiation, and thus reach the lower third of the central and parietal regions, where they are perceived. It is, as yet, impossible to distinguish between the course of tactile, painful, and muscular impressions from the face to the cortex of the brain.

C.—Sensory impressions from the great cavities of the body enter the spinal cord by the posterior nerve-roots, and are probably received by sensory cells which lie in the inner

and posterior part of the gray matter of the spinal cord—the Clarke column of cells. From these cells, these impressions pass outward to a white column lying in the lateral periphery of the cord—the direct cerebellar column—in which they pass upward to the medulla, and on through the restiform body to the cerebellum, to terminate either in the nucleus dentatus or in the cortex, or in both. The existence of a decussation of these impulses is undetermined, but, if it occurs, it must take place soon after their entrance, as each direct cerebellar column passes to that hemisphere of the cerebellum of the same side upon which it lies. The impressions thus transmitted to the cerebellum are not those of touch, pain, temperature, or the muscular sense, and are probably impressions connected with the functions of vegetative life and destined to awaken reflex actions.

If the facts here presented prove of value in aiding the diagnosis of local foci of disease in the central nervous system, the object of the author will be attained.

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